This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

**Usage guidelines**

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.

+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.

+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.

+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

**About Google Book Search**

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`

LANE MEDICAL LIBRARY STANFORD
2 45 0173 1154

*With the Compliments of*

*Dr. CHARLES PHELPS,*

# The Differential Diagnosis of Traumatic Intracranial Lesions.

BY

CHARLES PHELPS, M. D.,

Surgeon to Bellevue and St. Vincent's Hospitals;
Consulting Surgeon to Gouverneur Hospital.

REPRINTED FROM THE
New York Medical Journal
*for Nov. 10, Dec. 8, 15, 22, 29, 1894,
and Jan. 5 and 12, 1895.*

M593
P53
1895

LANE

MEDICAL LIBRARY

SEAL OF THE LELAND STANFORD JUNIOR UNIVERSITY
SEMPER VIRENS
1885

LEVI COOPER LANE FUND

GAYLORD BROS
MAKERS
SYRACUSE, - N.Y.
PAT. JAN. 21, 1908

593
53
895

*Reprinted from the New York Medical Journal.*

LANE LIBRARY

LANE MEDICAL LIBRARY
15677
SAN FRANCISCO

# THE DIFFERENTIAL DIAGNOSIS OF TRAUMATIC INTRACRANIAL LESIONS.*

BY CHARLES PHELPS, M. D.,
SURGEON TO BELLEVUE AND ST. VINCENT'S HOSPITALS;
CONSULTING SURGEON TO GOUVERNEUR HOSPITAL.

IN a paper read before this association two years ago, I indicated the comparative facility with which it is possible to diagnosticate cerebral traumatisms from morbid conditions of internal origin, and also recognized the difficulty of discriminating various encephalic injuries from each other.† Since that time additional observations and analyses of their results have served to broaden the diagnostic horizon. I submitted then an abstract of one hundred and twenty four cases. I can now add to these one hundred and seventy six others which I have since noted, a total of three hundred, of which one hundred and thirty terminated in death and were made the subject of necropsy.

---

* Read before the New York State Medical Association, October 9, 1894.

† A Clinico-pathological Study of Injuries of the Head. *New York Med. Jour.*, January 14, 21, 28, 1892.

COPYRIGHT, 1894, BY D. APPLETON AND COMPANY.

These additional cases may be summarized as were those previously recorded:

I. *Fractures involving the base:*

| | |
|---|---|
| Recovered | 36 |
| Died | 59 |
| Number of necropsies | 52 |

II. *Fractures confined to the vertex:*

| | |
|---|---|
| Recovered | 31 |
| Died | 12 |
| Number of necropsies | 7 |

III. *Intracranial injuries without fracture:*

| | |
|---|---|
| Recovered | 21 |
| Died | 17 |
| Number of necropsies | 13 |

The consolidation of the two series affords an aggregate of:

I. *Fractures involving the base:*

| | |
|---|---|
| Recovered | 57 |
| Died | 108 |
| Number of necropsies | 87 |

II. *Fractures confined to the vertex:*

| | |
|---|---|
| Recovered | 52 |
| Died | 22 |
| Number of necropsies | 17 |

III. *Intracranial injuries without fracture:*

| | |
|---|---|
| Recovered | 28 |
| Died | 33 |
| Number of necropsies | 26 |

| | |
|---|---|
| Total number of recoveries | 137 |
| " " " deaths | 163 |
| " " " necropsies | 130 |

In accordance with a more restricted purpose I shall confine myself primarily to the abstraction of essential symptoms in the fatal cases, and to their attendant lesions as verified by necropsic inspection. In order to make this synopsis complete in itself, I shall reproduce in connection with these recent histories an epitome of the analogous cases included in the former series.

The temperatures quoted are rectal; and hæmorrhage is denominated cortical when derived from laceration of the surface of the brain, pial when occasioned by rupture of the pial vessels in meningeal contusion, and epidural when situated between the dura and the skull.

### FRACTURES INVOLVING THE BASE.

Case I. *Symptoms.*—Wild delirium; high temperature; coma; hæmorrhage from left ear; death in forty-eight hours.

*Lesions.*—Laceration of both parietal lobes upon their lateral and inferior surfaces; cortical hæmorrhage.

Case II. *Symptoms.*—Active delirium only noted; walking case. Suicide by drowning after twenty-four hours.

Case III. *Symptoms.*—Coma, stertor, general muscular rigidity, and dilatation of left pupil; hæmorrhage from right ear, and later from right nostril; left hemiplegia after twelve hours, with disappearance of earlier symptoms; urine not conrolled; consciousness not restored. Death in six days.

*Lesions.*—Laceration of inferior and external surfaces of left frontal and of right temporo-sphenoidal lobes; corresponding cortical hæmorrhages thinning toward base and vertex; general hyperæmia.

Case IV. *Symptoms.*—Semicoma, mild delirium, imperfect articulation, with slow and irregular respiration, which continued for three days; paralysis of right upper extremity and of right upper and lower face, differing in degree at different times; mental condition varying from normal to one of noisy delirium; patient usually restless and during last two days unconscious. Temperature on admission, 101°; during first week, 100°; in second week, 99°+; and rose steadily from 103° to 109° through last two days. Death in forty-five days.

*Lesions.*—Su [illegible] neath the site of depressed fracture, external to right parietal eminence; subarachnoid serous effusion; general hyperæmia.

CASE V. *Symptoms.* — Stupor; gradual unconsciousness; delirium requiring mechanical restraint. Temperature on admission, 102°; ten hours later, 105°; declined to 101·8°, and then rose steadily to 106·6°. Death in twenty-nine hours.

*Lesions.*—Laceration of inferior surface of left frontal and of both temporo-sphenoidal lobes, and of inner border of right frontal lobe; pial hæmorrhage over upper surface of both hemispheres; general hyperæmia.

CASE VI. *Symptoms.*—Stupor; incoherence; dilatation of left pupil; slight deviation of eyes to the right; second day—delirium, muscular tremor, irregular pupils, frequent and intermittent pulse. Temperature on admission, 103°; five hours later, 102°; and rose to 106·2°. Death in twenty-four hours.

*Lesions.*—Laceration of inferior surface of left frontal and temporo-sphenoidal lobes.

CASE VII. *Symptoms.*—Stupor; irregular pupils; general muscular rigidity; ataxic gait; diminished sensibility, and loss of urinary control. On the fourth day, temperature normal, mind clear, and muscular rigidity lessened; copious serous discharge from right ear and right facial paralysis; on the fifth day, increased muscular rigidity and recurrence of stupor; on the sixth day, unconsciousness and frequent general convulsions. Temperature on admission, twenty four hours after injury, 99°+; on the fourth day, normal; on the fifth day, 100°+; on the seventh day, 105°. Death on the seventh day.

*Lesions.*—Contusion of surface of right occipital lobe posteriorly, with minute hæmorrhages and softening; laceration of inferior surface of both frontal lobes, and of right cerebellum; epidural hæmorrhage over both occipital lobes beneath the site of an extensive depressed fracture; cortical hæmorrhage over inferior surface of cerebellum and beneath the situation of the epidural hæmorrhage, where it was firmly coagulated and surrounded by plastic inflammatory exudation; thrombi in superior longitudinal sinus and torcular Herophili, and in right lateral and inferior petrosal sinuses and jugular vein; subarachnoid serous effusion over parietal and occipital lobes.

Case VIII. *Symptoms.*—Coma; stertor; muscular rigidity of both sides, most marked on the right; strong contraction of both pupils, but more marked in the left; no change in general condition till death, fifty-four hours later. Temperature on admission, 100·4°, rising progressively to 103·8°; declined post mortem.

*Lesions.*—Laceration of anterior border of left temporo-sphenoidal lobe and of the anterior and internal borders of both frontal lobes; cortical hæmorrhage covering the whole base of the brain; subcortical laceration with clot occupying the whole interior of the left frontal and temporo-sphenoidal lobes, and filling with blood both lateral ventricles and both occipital lobes; slight epidural hæmorrhage at point where fracture began in right inferior and posterior parietal region; slight subarachnoid serous effusion; thrombus extending from torcular Herophili through right occipital and inferior petrosal sinuses into the jugular vein.

Case IX. *Symptoms*—Partial consciousness for twenty-four hours; became complete; hæmorrhage from both nostrils and from right ear; delirium on the fifth day with post-cervical muscular rigidity, restlessness, and retraction of the abdomen; Cheyne-Stokes respiration and death. Temperature for three days, 99·2°; on the fourth day, 103·2°; on the fifth day, 104·8°.

*Lesions.*—Laceration of antero-inferior border of left frontal lobe and of inferior surface of right cerebellum; cortical hæmorrhage covering superior and external surface of left cerebrum; general hyperæmia of the left hemisphere with punctate extravasations.

Case X. *Symptoms.*—Partial unconsciousness and left hemiplegia, followed by irritability; hæmorrhage from left nostril. Death in twenty-four hours.

*Lesions.*—Epidural hæmorrhage over left temporal region beneath a separation of the coronal suture; laceration of the right frontal, right parietal about the fissure of Rolando, and of left temporo-sphenoidal lobe.

Case XI. *Symptoms.*—Coma; stertor; pulse 70, respiration

7; dilatation of left pupil and contraction of right; paraplegia; hæmorrhage from left ear and nose and under left conjunctiva. Death in five minutes after admission.

*Lesions.*—Large pial hæmorrhage, mainly at the base; blood still fluid; slight lacerations of inferior surface of left frontal and temporo-sphenoidal lobes; slight cortical hæmorrhage from frontal laceration.

Case XII. *Symptoms.*—Right hemiplegia; lack of control of urine and fæces; slight hæmorrhage from nose and left ear; temperature on admission, 99·8°. Death in two days.

*Lesions.*—Laceration of left parietal lobe beneath parietal eminence.

Case XIII. *Symptoms.*—Coma; stertor; pupils, pulse, and respiration normal. Death in five days.

*Lesions.*—Pial hæmorrhage over both cerebra; laceration of inferior surface of left temporo-sphenoidal lobe.

Case XIV. *Symptoms.*—Patient momentarily unconscious, then walked to the hospital in a dazed condition, and afterward walked home. Severe cephalalgia for three hours, followed by gradual supervention of coma, which became complete in four hours. Death in eight hours.

*Lesions.*—Large pial hæmorrhage over external aspect of left frontal and parietal lobes; slight epidural hæmorrhage beneath fine linear fracture upon right side; slight contusions of brain substance.

Case XV. *Symptoms.*—Coma; dilatation of right and contraction of left pupil; right hemiplegia; pulse became slower and respiration more labored. Death in nine hours and a half.

*Lesions.*—Large epidural hæmorrhage from rupture of left middle meningeal artery; slight laceration of left parietal, and of lateral border of right temporo-sphenoidal lobe.

Case XVI. *Symptoms.*—Unconsciousness followed by stupor; slight but increasing dilatation of left pupil; slight hæmorrhage from left ear; rigidity of left side; labored respiration; later, complete left hemiplegia; temperature, 101·2°. Death in twenty-one hours.

*Lesions.*—Laceration of inferior and lateral surfaces of right temporo-sphenoidal lobe and slighter lacerations of inferior surface of frontal lobes along the median fissure; extensive cortical hæmorrhage over right cerebrum; general hyperæmia.

CASE XVII. *Symptoms.*—Unconsciousness; right hemiplegia; dilatation of right and contraction of left pupil; full and slow respiration; normal pulse; supervention of stertor; death in eleven hours

*Lesions.*—Extensive epidural hæmorrhage from rupture of left middle meningeal artery; general hyperæmia, with minute coagula and punctate extravasations.

CASE XVIII. *Symptoms.*—Unconsciousness for thirty minutes and subsequent irritability; hæmorrhage from right ear; second day, somnolence, continued irritability, and lack of urinary control, and temperature 102°; third day, delirium, temperature rising to 105·4°; fourth day, moderate dilatation of the pupils, restlessness, hyperæsthesia, increase of surface temperature, followed by deep coma; temperature, 104°+ in the morning and 105°+ in the evening till death, at the end of the seventh day, when it rose to 106·5°.

*Lesions.*—Deep laceration of lateral border of left temporo-sphenoidal lobe; cortical hæmorrhage over left occipito parietal region; cavity in right parietal lobe beneath point of fracture and opening upon the surface; subarachnoid exudation of thick pus over right posterior parietal and occipital lobes and in right inferior occipital fossa; general hyperæmia and punctate extravasations.

CASE XIX. *Symptoms.*—Permanent unconsciousness; irritability when disturbed; dilatation of both pupils, especially marked in the right; profuse hæmorrhage from left ear, which continued for twenty-four hours, and was then followed by serous discharge; general convulsive movements, most pronounced in the right leg; temperature, 100°; pulse, 80; single general convulsion, most violent on the left side, on the second day, and repeated on the third day; temperature rose steadily to 107·2°. Death in three days six hours.

*Lesions.*—Large epidural clot in left middle fossa; large and deep laceration of the lateral surface of left temporo-sphenoidal and of lateral and inferior surfaces of right temporo-sphenoidal lobes; small and deep laceration at right parieto-occipital junction; large cortical clot in left middle fossa; thin cortical coagulum over right cerebrum.

Case XX. *Symptoms.*—Unconsciousness and muttering incoherence when disturbed; subconjunctival hæmorrhage at outer part of left eye; slight temporary rigidity of right arm; restlessness and irritability; little change till death—in seven days ten hours. Temperature on admission, 101°; in two days rose to 104·8°, declined from fourth to sixth days to 101°+ to 102°+, and then rose progressively to 107°+.

*Lesions.*—Subcortical laceration of left frontal lobe, completely excavating and filling its interior with clot and disintegrated brain tissue and inclosed by a thin layer of cortex, except at the base, where it was covered only by the meninges; laceration of anterior two thirds of external border of right cerebellum; extravasation of blood of the size of a robin shot in the center of the right corpus striatum; slight cortical hæmorrhage over posterior part of the right cerebrum.

Case XXI. *Symptoms.*—Unconsciousness and delirium; hæmorrhage from mouth and nose; mental condition in twelve hours became normal; slow pulse; labored respiration; rigidity of arms, especially of the right; normal pupils, followed by restlessness, muttering delirium, lack of urinary control, and unconsciousness; temperature, 104·8° to 106°. Death in twenty-four hours.

*Lesions.*—Laceration of superior surface of both frontal and both parietal lobes.

Case XXII. *Symptoms.*—Coma; stertor; full pulse; pulmonary œdema beginning before admission; hæmorrhage from right ear; death in two hours.

*Lesions.*—Epidural hæmorrhage over both hemispheres and another of larger amount in the inferior occipital fossæ compressing the pons and medulla; slight lacerations of inferior surface of both frontal and right temporo-sphenoidal lobes with some cortical hæmorrhage.

Case XXIII. *Symptoms.*—Unconsciousness; restlessness; coma nearly complete on fourth day; pupils, pulse, and respiration normal; temperature on fourth day, 102·2°; on fifth day, 103° to 106°; on sixth day, 107°. Death in five days and a half.

*Lesions.*—Laceration of external surface of left frontal and left temporo-sphenoidal lobes, with consequent cortical hæmorrhage.

CASE XXIV. *Symptoms.*—Unconsciousness; persistent vomiting; hæmorrhage from left ear; moderate dilatation of pupils, especially of the left; temperature, 98·7°; second day, restoration of consciousness, delirium; third day, wide dilatation of both pupils, which were only slightly responsive to light, the left continuing to be more markedly affected than the right, and this condition permanent; sixth day, mind clear but apathetic, followed by stupor, with paresis and anæsthesia of all the extremities, more marked on the right side; later, left foot and right hand less paretic, mind clear, but senses blunted, some right facial paralysis, cephalalgia, rapid and feeble pulse; ninth day, delirium and recurring unconsciousness; eleventh day, patient neither spoke nor moved, nor was conscious of pain or external irritation; on the fifteenth day, death. Temperature below 100° till fourth day, when it rose to 103°, and then varied from 100° to 102° till the twelfth day, when it rose to 104°, and was afterward from 103° to 104·5°.

*Lesions.*—General hyperæmia with minute coagula and excessive œdema; slight lacerations of inferior surface of right temporo-sphenoidal lobe and one somewhat larger upon its external surface; very moderate cortical hæmorrhage extending over right occipital lobe.

CASE XXV. *Symptoms.*—Stupor; hæmorrhage from right ear; lack of control of urine and fæces; condition alcoholic; second day, active delirium, muscular tremor, delusions, and intervals of unconsciousness; sixth day, coma, stertor, muttering delirium, general muscular rigidity, slight contraction ot right pupil, and slight right facial paralysis; eighth day, two slight convulsions involving arms, face, and eyes, followed by paralysis of right arm and face, and elevation of surface temperature of left side; right side normal, left side 102°. Death on the eighth day. Temperature till fourth day, 100° to 102°; afterward 103° to 104° till eighth day, when it rose to 105·6° and declined to 104·8°.

*Lesions.*—Thin layer of pial hæmorrhage, covering both parietal and both occipital lobes, and meningeal hyperæmia; large subarachnoid serous effusion; general œdema of brain substance and minute vessels filled with coagula; fluid blood in anterior cornu of left lateral ventricle; small lacerations of superior and external surface of right frontal and of left occipital lobes and on either side of median fissure of cerebellum.

Case XXVI. *Symptoms.*—Vertigo and feeling of illness; hæmorrhage from right ear and nostril; temperature, 100°; soon followed by hæmatemesis, coma, and stertor. Death in four hours.

*Lesions.*—Epidural hæmorrhage, compressing right frontal lobe laterally; slight pial hæmorrhage on either side of the anterior columns of the medulla; extravasations into the pons in both transverse and longitudinal fibers, the largest a half by a quarter inch in diameter; general hyperæmia with punctate extravasations and coagula in the minute vessels.

Case XXVII. *Symptoms.*—Coma; stertor; frequent weak and irregular pulse; slight hæmorrhage from right nostril; protrusion of both eyes and dilatation of both pupils, especially of the left; rigidity of right side. Death in eight to ten hours.

*Lesions.*—Epidural hæmorrhage in right occipital fossa; small laceration of inferior surface of left frontal lobe anteriorly; cortical hæmorrhage over both frontal lobes.

Case XXVIII. *Symptoms.*—Paresis of left lower extremity; temperature, 98·8°; fourth day, temperature suddenly rose to 99°+ to 102°; fifth day, delirium, temperature 105·2° to 106°; death.

*Lesions.*—Laceration of inferior surface of left temporo-sphenoidal lobe; cortical hæmorrhage, filling left middle fossa.

Case XXIX. *Symptoms.*—Coma; stertor; dilatation of right pupil. left invisible from ecchymosis; anæsthesia followed by paralysis of right upper extremity; pulmonary œdema; second day, urinary control lost, mind clear, pupils normal, motor and sensory function restored; fifth day, gradual increase of temperature, subconjunctival hæmorrhage noted; sixth and seventh days, mental condition apathetic, subconjunctival hæmorrhage increased; eighth day, sudden unconsciousness, and on the ninth day, death. Temperature on admission, 101·6°; fifth day, 103°; sixth day gradually declined to 100°; eighth day, 104·8°, and rapid decline to 102·8°; ninth day, 106°.

*Lesions.*—Subcortical laceration, disintegrating and filling with clot the interior of both frontal lobes; on left side the median surface ruptured through the arm center and gyrus fornicatus, and the lateral ventricle invaded; deep laceration of external border of left cerebellum; internal brain structure softened and reddened in patches of limited contusion.

Case XXX. *Symptoms.*—Unconsciousness which continued till death; slight hæmorrhage from both nostrils; left pupil dilated; right contracted; loss of control of urine and fæces; face flushed; on the last day of life general sensation markedly diminished. Temperature on admission, 101°; till next day 101°+, then rose progressively to 106·8°. Death in three days and a half.

*Lesions.*—Deep and wide laceration across inferior surface of left cerebellum; laceration excavating inferior surface of right frontal, extending into temporo-sphenoidal, lobe; laceration of middle portion of gyrus fornicatus, half an inch in diameter; anterior fossæ filled with firm clot, three fluidounces by measurement; cortical hæmorrhage in right posterior fossa around foramen magnum, and also over right frontal lobe; general contusion of both hemispheres, most marked posteriorly.

Case XXXI. *Symptoms.*—Brief unconsciousness, which recurred in the ambulance; in the interval no evidence of serious injury; on admission, pupils contracted, sudden cyanosis, and death in twenty-five minutes.

*Lesions.*—Epidural hæmorrhage over left occipital lobe; pial hæmorrhage in inferior occipital fossæ, compressing the medulla; cortical hæmorrhage over both frontal and both temporo-sphenoidal lobes from laceration of their inferior surfaces.

Case XXXII. *Symptoms.—Primary:* Consciousness retained; hæmorrhage from right ear; no general symptoms; temperature, 100·4°, and afterward normal. *Secondary:* On the twentieth day, some lethargy and frontal headache; twenty-third day, severe frontal headache, delirium, somnolence, and left hemiplegia; twenty-fourth day, complete left hemiplegia and hemianæsthesia, insensibility of both pupils, continued somnolence, normal mental condition when roused, temperature 98·5°, pulse 56, cicatrix of wound firm and uninflamed; twenty-fifth day, patient roused with difficulty, action of bladder and rectum unconscious and involuntary, temperature 100·2°; operation and evacuation of pus, one to two drachms, from subcortical abscess, beneath the angular gyrus. Temperature rose to 108°, and death occurred sixteen hours later.

In the interval between the primary and secondary symptoms there was posterior cervical glandular enlargement, with some constitutional reaction.

*Lesions.*—Subcortical abscess cavity, which had been evacuated during life. No superficial laceration or hæmorrhage.

Case XXXIII. *Symptoms.*—Unconsciousness, which continued till death; hæmorrhage from left ear; dilatation of both pupils, and subsequent contraction of the right; muscular relaxation, followed by later rigidity; temperature on admission, 99·6°; afterward 99·6° to 100·4°; an hour post mortem, 101·2°. Death in twelve hours.

*Lesions.*—Deep laceration of posterior portion and inferior surface of left temporo-sphenoidal lobe, with consequent cortical hæmorrhage over occipital lobe; excessive cerebral hyperæmia.

Case XXXIV. *Symptoms.*—Coma; stertor; loss of urinary control; hæmorrhage from nose and later hæmatemesis; pulse, 96 and full; respiration, 18; temperature, 100°, rising gradually to 102·6° some time before death, in fourteen hours after admission.

*Lesions.*—Small epidural hæmorrhage at site of fracture; rupture of dura; corresponding laceration in anterior inferior parietal region; laceration of anterior half of right middle temporal convolution; small laceration in center of left cerebellum filled with fluid blood; general hyperæmia, most marked on left side posteriorly.

Case XXXV. *Symptoms.*—Unconsciousness; contraction of both pupils; rigidity of both lower and of right upper extremities; pulse and respiration too rapid to be counted; temperature, 101°, and in articulo mortis, 100·4°. Death in two hours. Temperature two hours post mortem, 99°+.

*Lesions.*—Pial hæmorrhage over left frontal and parietal lobes, superiorly and externally, and over region of right occipito-parietal junction; subcortical laceration of left corpus striatum at junction of middle and posterior thirds; excessive general hyperæmia.

Case XXXVI. *Symptoms.*—Coma; stertor; alcoholic condition; no superficial injury; muscular relaxation; face flushed; pupils slightly contracted; vomiting; temperature, 97°, continued to be subnormal; pulse, 60; respiration, 16; one general convulsion just before death, at the end of eight hours and a half.

*Lesions.*—Fracture through left occipital, parietal, and squamous portion of temporal bone to margin of petrous portion; laceration of inferior surface of right frontal lobe and both temporo-sphenoidal lobes; pial hæmorrhage over whole right parietal region.

Case XXXVII. *Symptoms.*—Consciousness lost but regained before admission after suicidal pistol-shot wound of the head; total loss of vision; exophthalmia of both eyes; dilatation of both pupils, which were irresponsive to light; temperature, 100·2°; pulse, 60; respiration, 20. Ophthalmic examination by Dr. P. A. Callan on the second day disclosed only patches of retinal hæmorrhage; mental condition unimpaired; sense of smell entirely lost. On the fourth day an unsuccessful attempt was made to extract the ball, and a drainage-tube was afterward carried from the foyer of entrance through both frontal lobes to a cranial opening made upon the opposite side; followed for five days by discharge of brain tissue, and then till death by pus in increasing quantity. Mental condition normal till the fifth day, sluggish till ninth day, and afterward marked by increasing delirium, which lapsed into a muttering stupor at the end of life on the thirteenth day. Temperature at time of operation, 99°; rose to 103·6° in ensuing twenty-four hours, and then varied from 102·4° to 104·5° on the last day; pulse and respiration nearly normal till just before death.

*Lesions.*—Ball penetrated external wall of the right orbit, just behind the external angular process; passed beneath optic nerve, comminuted inner wall of the orbit, crista galli, cribriform plate, and lesser wing of the sphenoid; entered left orbit through inner wall, and was found beneath left optic nerve. In the cranial cavity its course was beneath both optic nerves. Subarachnoid purulent effusion covered both frontal lobes, more copious on the left side and at the base; left frontal lobe was excavated and filled with pus and brain detritus; pus also existed in the tract of the drainage-tube through the right frontal lobe.

I am indebted to a colleague for the opportunity of observing this case.

Case XXXVIII. *Symptoms.*—The patient, after having passed through three hospitals, with three discharges and two transfers, and after having wandered about the streets and suffered much exposure, was finally received and allowed to remain in an asylum for the insane on the eighth day after a fall from the stoop of her house. She had then delusions and other symptoms of mental derangement, left facial paralysis, left subconjunctival hæmorrhage, and hæmorrhage from both ears. She died on the twenty-fourth day from the reception of the injury.

*Lesions.*—Transverse fracture of the base, extending through both petrous portions and left orbital plate; laceration of inferior surface of left frontal lobe; small subcortical laceration of left parietal lobe; cortical hæmorrhage at base and over the external surface of both hemispheres; general contusion.

Case XXXIX. *Symptoms.*—Profound coma, which continued till death; stertor; pulse, 70, full and strong; temperature, 99·4°. Death in seven hours.

*Lesions.*—Linear fracture through right side of the occipital bone to jugular foramen; pial hæmorrhage over both occipital and posterior portion of left parietal lobes; excessive general hyperæmia.

Case XL. *Symptoms.*—Contusion of left parietal region; primary unconsciousness; epistaxis; delirium, which continued till admission to hospital two days later; unconsciousness at that time; pupils normal; pulse rapid and weak; respiration, 21; temperature, 101·4°, rose to 102°+; consciousness not restored. Death in four days.

*Lesions.*—Linear fracture of occipital bone from tuber to right jugular foramen; also fissure of left orbital plate; thrombosis of lateral sinuses; clot firm, but not decolorized; general hyperæmia, with a few minute coagula.

Case XLI. *Symptoms.*—Unconsciousness succeeding an injury received on the preceding day; admission to hospital after twenty-four hours; right pupil slightly dilated; temperature, 99·8°; pulse, 96; respiration, 24; temperature rose to 100·2°. Death in about thirty hours from time of injury.

*Lesions.* — Linear fracture running nearly transversely through left parietal bone into right coronal suture; also V-shaped fracture from *contre-coup* in right middle fossa; epidural hæmorrhage over right frontal lobe from vertex to base; laceration of middle two fourths of second right temporal convolution, with cortical hæmorrhage extending over parietal lobe; general hyperæmia with minute coagula in all parts of the brain.

Case XLII. *Symptoms.*—Shock; consciousness retained; temperature, 96°; pulse, 78; respiration, 21; sudden cyanosis, with extreme dyspnœa, and loss of consciousness which lasted for only three or four minutes, followed by numbness of both arms; no further dyspnœa; delirium fourteen hours later, and death four hours later still, preceded by a single convulsive movement and without respiratory disturbance.

*Lesions.*—Occipital contusion and wound behind right ear; bifurcated linear fracture in right inferior occipital fossa; pial hæmorrhage beneath tentorium, extending around lateral borders of cerebellum and covering the pons. Moderate general hyperæmia.

Case XLIII. *Symptoms.*—Scalp wounds in left parietal and large hæmatoma in right parietal region; compound linear left parietal fracture; no known loss of consciousness; shock; dilatation of both pupils; pulse feeble; respiration shallow; temperature after four hours, 96·4°. Death in nine hours and a half.

*Lesions.*—Parietal fissure extended nearly across greater wing of sphenoid; considerable subarachnoid serous effusion; general hyperæmia and thrombosis of minute vessels, most marked posteriorly.

Case XLIV. *Symptoms.*—Unconsciousness till death; pupils normal; muscular twitchings over whole right side of the body; temperature on admission, 98°; in six hours, 103·6°; in seven hours, 104·4°; in nine hours, 106·6°; pulse, 80 to 145; respiration, 15 to 34. Death in nine hours and a half.

*Lesions.*—Calvarium crushed; large wound in the skull at the vertex involving the median line, two by three inches in its diameters; on the left side the osseous fragments rested upon the dura, on the right they deeply penetrated the brain; a fissure extended into the right orbital plate; epidural clot on the left side in which the parietal fragments were imbedded; on the right side, disintegrated brain tissue, bone, and membranes were commingled; anterior part of both lateral ventricles contained blood; cortical hæmorrhage extended beneath the tentorium; general hyperæmia and vessels even of larger size filled with thrombi.

Case XLV. *Symptoms.*—Scalp wound in left frontal region; left pupil dilated; consciousness only partially lost; temperature on admission, 98°; fell in four hours to 97·6°; pulse, 90; respiration, 24. Death in four hours and a half.

*Lesions.*—Slight depression at left external angular process of frontal bone and fissure, extending thence through both orbital plates and intervening ethmoid body; deep laceration of frontal lobes on either side of inferior median fissure; smaller laceration of posterior border of cerebellum, near median line, from which a cortical hæmorrhage extended over both its superior and inferior surfaces; general hyperæmia and minute coagula.

Case XLVI. *Symptoms.*—Hæmatoma over whole vertex, and small wound of the scalp; unconsciousness which continued till death; dilatation of left pupil; general convulsions, beginning in the hands, with marked opisthotonos; temperature six hours after reception of injury, 98·6°; pulse, 84; respiration, 28; extent of fracture determined by incision. Death in nine hours.

*Lesions.*—Disjunction of coronal suture, multiple fissure of frontal bone, and fissure through right parietal and occipital bones, with branch into posterior fossa; laceration of right frontal, parietal, and occipital lobes, and wound of dura permitting escape of brain tissue through the osseous parietal opening.

Case XLVII. *Symptoms.*—Contusions of left side of head and face and tactile evidence of simple fractures; unconsciousness which continued till death; epistaxis and hæmatemesis; temperature on admission, 99°; pulse, 96; two hours later—temperature, 96·4°; pulse, 140; respiration, 53; five hours later—temperature, 95·6°; pulse and respiration as before; second day, deglutition became possible and sensitiveness to external impressions was regained; pupils slightly dilated; temperature, 103° to 103·6°; pulse, 168 to 196; respiration, 48 to 58. Death in thirty-four hours. (In this, the case of a child, four years and a half of age, the brain weighed forty-eight ounces, and was in all respects symmetrical; the skull was of normal thickness.)

*Lesions.*—Separation of the coronal and of the bifrontal suture to nasal bones, which were fractured; fracture continuous into ethmoid body, with complete detachment of the crista galli and cribriform plate; fissure of right parietal bone and depressed fracture of left frontal above orbital ridge; slight epidural hæmorrhage over the vertex; laceration of frontal lobes in the space corresponding to the site of cribriform plate; general hyperæmia with minute coagula, most marked in cerebellum and occipital lobes.

Case XLVIII. *Symptoms.*—None discovered, and admission to hospital refused two days after a fall in the street; found dead two hours later a block away; wound over left eye.

*Lesions.*—Pneumonia involving lower lobe of right lung, and large, flabby heart; fracture extending through left supraorbital ridge and orbital plate into greater wing of sphenoid bone; general hyperæmia and thrombosis.

Case XLIX. *Symptoms.*—Large hæmatoma over right eye; profuse hæmorrhage from mouth, nose, and right ear; unconsciousness; rapid and feeble pulse and respiration; dilatation of both pupils, especially the left. Death in fifteen minutes. (Cæsarean section at six months and a half; child lived forty-five minutes.)

*Lesions.*—Separation of right *sutura additamentum lambdoidalis* and fissure continued, through petrous portion and middle fossa, into body of sphenoid bone; large pial hæmorrhage over left parieto-occipital region.

Case L. *Symptoms.*—Wounds over left eye and at the occiput; shock; unconsciousness; hæmorrhage from ears, nose, and mouth; restlessness, and utterance of short, sharp cries; pulse frequent, weak, and symmetrical; respiration slow, irregular, and sighing; right pupil dilated, left invisible from ecchymosis; twitching of right side of face, followed by general convulsions, preceded death at end of twelve hours.

*Lesions* discovered by incisions: In left temporal region fissures ran into temporal fossa, and squamous suture was partially disrupted; in occipital region open fissure ran into right petrous portion and lambdoid suture was separated; arachnoid hæmorrhage in right occipito-parietal region.

Case LI. *Symptoms.*—Scalp wounds in parietal regions; mobility and crepitation of calvarium; shock; unconsciousness which continued till death; slight epistaxis and profuse hæmatemesis; both pupils dilated, and after three hours and a half the right more so than the left; one radial pulse fuller and stronger than the other; temperature on admission, 101°; in one hour, 102°; in two hours, 106°; in four hours, 106·8°; pulse, 70, 110, 160, 170; respiration in two hours, 48. Death in four hours and a half.

*Lesions.*—Fissure, beginning just above left internal angular process, running across middle of parietal bones, and nearly circumscribing the calvarium; another detached its posterior portion, and others still extended from the primary line of fracture to the base; arachnoid hæmorrhage on the left side; further examination refused.

Case LII. *Symptoms.*—Contusion in left frontal region and ecchymosis of left eye; consciousness retained; hæmorrhage from right ear and from nose and mouth; delirium, becoming violent later in the day and during the night; on the second day, the patient formed a fixed delusion that he had fallen from a mulberry tree. He described with circumstantiality all the details of his imaginary accident. He had no recollection of the manner in which his injury had really occurred, and would give no credence to facts as they were presented to him; he had other and transitory delusions, but this one remained unalterable. Both pupils were moderately and symmetrically dilated. His mind became remarkably alert, and his conversation was logical and coherent. Nine days later hæmorrhage from the right ear recurred, subconjunctival hæmorrhage, which had been previously noted, increased, and the left eye became prominent. Mechanical restraint was still necessary to keep him in bed. On the twelfth day, hæmorrhage from the ear ceased, and subconjunctival hæmorrhage diminished; a scarcely perceptible facial paralysis existed. His mind seemed clearer; he could recollect the street and neighborhood in which he lived, but not the number of his house; only the one delusion persisted. Later, a frontal headache from which he had constantly suffered became less urgent; but his general condition was not materially changed till two days before his death, when he became progressively asthenic from an intercurrent diarrhœa. His mind remained clear with occasional transient delusions, his conversation coherent, and his belief in the mulberry tree unshaken to the last. Temperature on admission was 98·6°; one hour later, 100°; and five hours later, 104·7°; for the two days following it was 103° to 103·8°; and during the fourth and fifth days, 101° to 99° +; it varied till the twentieth day from 99·8° to 101·8°, only twice exceeding 100°. The pulse on admission was 85, and the respiration 20, with no considerable subsequent changes till near the close of life. Thirty-six hours ante mortem temperature rose to 102·4°, and twelve hours later to 105°; in another twelve hours it declined to 97·5°, and immediately before death rose again to 100°, with pulse 140, and respiration 42. Death on the twenty-fourth day.

*Lesions.*—Depressed fracture above left supraorbital ridge with fissure extending across both orbital plates and intervening cribriform plate, through right middle fossa, external to greater wing of sphenoid, through outer part of petrous portion of temporal into posterior fossa, and returning upon itself across the petrous portion and through the body of the sphenoid and ethmoid to finally terminate in itself anteriorly. A second fissure crossed the left orbital plate into the left middle fossa. Small epidural hæmorrhage beneath the depressed part of the fracture; laceration of under surface of both frontal lobes, mainly subcortical, crossing median line obliquely from center of left lobe to line of right anterior cerebral artery. This laceration was an inch and a quarter wide by an inch in depth at its commencement on the left side, and on the right side was five eighths of an inch in width by half an inch in depth. In removing the brain the arachnoid was torn and the diffluent contents of the cavity escaped; its margin and the overhanging cortical tissue were dark and sloughy; its deeper portion was yellow and ragged; it was separated anteriorly on the left side from the median fissure by a single convolution. There were general hyperæmia and minute thromboses, most marked posteriorly.

Case LIII. *Symptoms.*—Hæmatoma in left parietal region; coma; stertor; no response to external irritation; pupils widely dilated; pulse full, slow, and strong; temperature on admission, 99°, and rose steadily to 107·8°; respiration, 32, 46, 14; pulse, 62, 70, 126. Death in four hours and three quarters.

*Lesions.*—Coronal suture separated and fracture continued into anterior part of middle fossa on both sides; general hyperæmia with well-marked but not excessive œdema, and some punctate extravasations.

Case LIV. *Symptoms.*—Consciousness lost but partially restored on arrival of ambulance; large hæmatoma in right posterior occipital region; slight epistaxis; pupils moderately contracted; respiration shallow; right radial pulse after two hours more frequent than the left—84 and 74, 114 and 110; temperature on admission, 96°; in two hours, 95°; in six hours normal, and rose to 100·4° before death in nine hours.

*Lesions.*—Separation of coronal suture on the left side and fracture continued through middle fossa, sella turcica, right middle fossa, right petrous portion, and posterior fossa, to foramen magnum; large epidural clot in left temporal region; slight cerebral œdema; old meningeal adhesions, and small white nodules in the pia.

CASE LV. *Symptoms.*—Consciousness retained for fifteen minutes after admission; then delirious four hours; afterward consciousness lost; contusion of right side of head; hæmorrhage from left ear and nose, and hæmatemesis; slight dilatation of right pupil; temperature on admission, 101°, and rose to 103°; pulse, 90 to 108; respiration, 22, 24. Death in ten hours.

*Lesions.*—Linear fracture extended from right squamous portion through body of sphenoid and both middle fossæ into left temporal bone; a second fissure extended from sphenoid into cribriform plate; large epidural clot in left middle fossa; marked general hyperæmia.

CASE LVI. *Symptoms.*—Delirium which continued till final unconsciousness at the close of life; wound in left temporal region; hæmorrhage from right ear; second day, paralysis of left arm; fourth day, loss of control of urine and fæces; and death in three days and eight hours; temperature on admission, 99°+; rose to 103·2°, declined to 101°, and rose to 106·6° shortly before death; pulse, 90 to 114; respiration, 18 to 28.

*Lesions.*—Linear fracture extending from outer part of right petrous portion, through body of sphenoid bone into its left lesser wing; epidural hæmorrhage in left middle fossa; large pial hæmorrhage over right temporal and parietal lobes, especially profuse near the vertex; general hyperæmia with minute coagula; thrombus in each lateral sinus.

CASE LVII. *Symptoms.*—Consciousness lost, partially recovered after admission; articulation imperfect; alcoholic condition; small wound in right occipital region; active delirium a few hours later; alternations of delirium and stupor till death, sixteen days afterward; temperature, pulse, and respiration normal from second to fourth day; temperature varied from 99·4° to 104·8° and was 103° at time of last observation; pulse, 112 to 144; respiration, 26 to 44.

*Lesions.*—Fracture extending from right of foramen magnum, three inches and a half, into the left inferior occipital fossa; laceration of inferior surface of both frontal and left temporo-sphenoidal lobes; pial hæmorrhage over right occipital lobe; general subarachnoid serous effusion.

CASE LVIII. *Symptoms.*—Coma which lasted for a few hours; wound in occipital region; no general symptoms noted till seventh day, when sudden recurrence of coma and death; temperature second, third, and fourth days, 100·4° to 99·4°; after second coma, 104°; pulse and respiration normal.

*Lesions.*—Fracture through right middle fossa, involving petrous portion; laceration of inferior surface of right frontal and temporo-sphenoidal lobes; arachnoid hæmorrhage over almost entire surface of right cerebrum; clot in the substance of the right centrum ovale.

Case LIX. *Symptoms.*—Momentary unconsciousness; contusion of left eye and wound of left frontal region; epistaxis without perceptible injury of the nose; second day, delirium at intervals, becoming constant through the night; third day, somnolence, restlessness, and delirium characterized by delusions; at close of the fourth day delirium became muttering and respiration stertorous. Death in four days and a half. Temperature on admission, 99·8°; on the second day, 104·8°, 102°, 100°, 103°, 101·8°; on the third day, 103·6°, 103·4°; on the fourth day, 103°+, 106·6°; on the fifth day, 107°, 108·2°. Pulse till end of fourth day, 82, 56, 90, 106; respiration, 19, 34, 24, 40.

*Lesions.*—Fracture beginning at left external angular process of frontal bone, comminuting orbital plate, extending into body of sphenoid, and, after bifurcation, terminating in the cribriform plate and in squamous portion of right temporal bone; two lacerations of inferior surface of left frontal lobe—one near its center as large as a hickory nut, containing disintegrated clot and brain tissue, the other smaller and more superficial, encroaching upon the middle portion of the Sylvian fissure; two other slight lacerations existed upon the inner border of right occipital lobe; slight subarachnoid serous effusion upon upper surface of cerebellum; general hyperæmia with some minute coagula.

Case LX. *Symptoms.*—Consciousness not lost, but delirium continuous from time of injury till final coma; contusion behind left ear; very slight dilatation of pupils; delirium became violent; at the end of two or three hours the patient became aphasic; he could utter single words correctly, or a number of words in succession, each correct in itself, but strung together without sense or logical sequence, as "water—father—when," or "Jesus—now—who." He also connected fragments of words with each other, as "en—is—other," meaning when is mother; or "J—mother," for Jesus, mother; or "J—ter," for Jesus, water; sometimes "ter—J," for water, Jesus. The clew to these fragmentary words and sentences was found in the words he constantly used singly. The aberrations of speech, like the delirium, continued till final coma, and were constant. On the second day his head was extended, but without cervical rigidity; he was restless and irritable; the pupils were still normal; urine was retained; coma and stertor supervened, and death occurred thirty-seven hours and a half after admission. The temperature on admission was 100·2°, rose progressively in twenty hours to 105·2°, remained stationary for twelve hours, and again rose progressively to 108·6°. One hour post mortem it was 110°. The pulse constantly increased in frequency from 90 to 190. The respiration did not exceed 24 for thirty-two hours, after which it was from 40 to 50.

*Lesions.*—Fracture extending from the left superior occipital fossa, through the posterior condyloid foramen, into the foramen magnum; epidural hæmorrhage, slight over the occipital lobes and more abundant in the inferior occipital fossæ; cortical hæmorrhage in central part of anterior fossæ and over sella turcica; thrombi in left lateral and superior petrosal sinuses; posterior meningeal veins, including those of larger size, greatly distended; some opacity of arachnoid and subarachnoid serous effusion over right occipital lobe. Lacerations were confined to the base, except in case of left temporo-sphenoidal lobe. The first left temporal convolution was lacerated through the whole thickness of its cortex for a length of an inch and a half, which included the second and part of its third fifths, estimated from its anterior extremity, and its middle portion involved the second convolution. This laceration was limited to the exact width of the two convolutions and was covered by the unruptured arachnoid. A small and deep laceration existed upon the inferior surface of this lobe, and another, small and shallower, was situated at its tip, involving all three of its convolutions. A similar slight laceration occurred at the anterior extremity of the right temporo-sphenoidal lobe, including the second and third convolutions. There was an extensive laceration of the under surface of the left frontal lobe, extending from its anterior border to the optic chiasm and from the median line outward through the first and second into the third orbital convolution; it disintegrated the cortex and the subcortex to a considerable depth, and the resultant hæmorrhage had broken through into the arachnoid cavity. There was finally a small contusion about the center of the inferior surface of the right frontal lobe. The brain substance was generally hyperæmic with minute thromboses, and a small amount of reddish serum occupied the lateral ventricles.

Case LXI. *Symptoms.*—No evidence of brain lesion on admission, twenty-four hours after injury, except right radial pulsation was fuller and stronger than the left; followed by delirium, with delusions, after sixteen hours, which, with the unsymmetrical pulse, persisted for five days. On the sixth day, mind clear, memory restored, general headache; later, delirium at intervals, aimless inclination to get out of bed, increasing difficulty of articulation, progressive mental impairment; control of bladder and rectum lost. On the twenty-fifth day, condition quiet, weaker, picking at the bedclothes. Twenty-sixth and twenty-seventh days, delirium, irritability, great sensitiveness to external disturbances, unconsciousness. Death occurred on the twenty-eighth day. Temperature on admission, 102°; second and third days, 103°, 104·4°; from this time it was usually 99° to 99°+, sometimes normal, occasionally 100°, until the last eighteen hours, when it suddenly rose to 105°, and, with slight recessions, finally reached 108°. Pulse, 84 to 54, till the last four days, when it exceeded 100; but in the last twelve hours, with the highest temperatures, it ranged from 70 to 54. Respiration was accelerated on the second and third days, but at other times was normal till within a few hours of death. Right axillary temperature the day before death was from 0·2° to 1·2° higher than the left. Temperature one hour post mortem, 108°.

*Lesions.*—Open fissure extended from beneath torcular Herophili, downward and slightly outward, to a point near the left margin of the foramen magnum, where it subdivided to inclose a quadrangular depression of bone three quarters of an inch by half an inch in its diameters; it was then continued between the posterior border of the petrous portion and the basilar process of the occipital bone, where it terminated. No external evidences of injury; small epidural hæmorrhage on either side of median line at commencement of fracture; two thin laminar spots of epidural clot, each about half an inch in diameter, firmly attached to the dura over anterior part of the left frontal lobe, from *contre-coup;* corresponding blood stains upon the surface of the bone, but under surface of dura not discolored; large subarachnoid serous effusion over the vertex; meningeal hyperæmia, but not of the cerebral surface. Four lacerations of the antero-superior surface of the left prefrontal lobe, with contusion of intermediate cortex, the whole covering a space an inch and a half in diameter; another laceration of small size a short distance behind them; small laceration upon the anterior part of the external surface of the right frontal lobe; these lacerations all extended into the subcortical substance and were partially filled with necrotic tissue; the adjacent brain substance was unaltered. Marked general œdema and hyperæmia, with moderate number of punctate extravasations and minute thrombi; brain of normal consistence.

Immediate microscopic examination afforded no evidence of inflammatory action, except in contiguity to the necrotic tissue. The quadrangular osseous depression was firmly fixed, but there was no osseous deposit.

Case LXII. *Symptoms.*—No history; walking case; semi-consciousness, but without speech or comprehension of speech then or afterward; hæmorrhage from left ear, and œdema of the left mastoid region; pupils normal; early delirium; sensitiveness to external irritation; retention of urine. Second day, entire unconsciousness; convulsive movements of the limbs; Cheyne Stokes respiration; accumulation of mucus in the trachea and bronchi, and death in forty-two hours. Temperature on admission, 101·6°; in twenty four hours, 103·2°; in twenty-seven hours and till death, 108·6°; one hour post mortem, 108°.

Pulse, 64 to 50; second day, 140 to 168. Respiration normal, 24, 16. 20.

*Lesions.*—Hæmatoma in left occipital region; linear fracture through left occipital bone, from median line and along the groove for the lateral sinus, across the petrous portion by a wide fissure, and separating the dorsum ephippii from the sphenoid bone; thrombus in left lateral sinus; complete disintegration of the right frontal lobe to within half an inch of the fissure of Sylvius and quite to the anterior border of the corpus striatum; deep laceration of greater part of the inferior surface of the left temporo-sphenoidal and a smaller laceration in the center of the inferior surface of the right temporo-sphenoidal lobe; cortical hæmorrhage from the frontal laceration filled all the basic fossæ except the outer part of the left anterior, and one clot in the median line anteriorly was as large as a mandarin orange; it also covered with a thin coagulum the superior and lateral surfaces of the whole right and greater part of the left hemispheres, and extended over the superior surface of the cerebellum; general hyperæmia, with a few minute coagula; minute extravasations in the center of the pons, the largest of which was about the size of a robin shot.

Case LXIII. *Symptoms.*—Primary unconsciousness, followed by mental hebetude and mild delirium, which continued till death; occasional dysphagia in second week, sometimes extreme. Temperature on admission, 99·2°, rose in two hours to 102°, and was afterward 99° to 100° and 101°. Pulse on admission, 50; below 90 for eight days; afterward exceeded 100. Respiration normal. Death occurred in fourteen days, and was immediately preceded by extreme dyspnœa and dysphagia.

*Lesions.*—Fracture through left occipital, from median line to petrous portion of temporal bone; extensive laceration of antero-superior and inferior surfaces of left frontal lobe; cortical hæmorrhage covered with a thin clot the entire left hemisphere and posterior half of the right, and filled all the basic fossæ.

Case LXIV. *Symptoms.*—Contusion in occipital region, and recurrent hæmorrhage from left ear; violent delirium after hirty-six hours; right radial pulse fuller and stronger than the left on the third and fourth days. Temperature on admission, 98·4°, rose in twelve hours to 102°, and afterward varied from 98·5° to 100° + in the morning, and from 99·5° to 100·8° in the evening; last observation, six hours ante mortem, 101·6°. Pulse and respiration were practically normal. Death in ten days.

*Lesions.*—Fracture through posterior part of left parietal into petrous portion of temporal bone; transverse laceration across inferior surface of right frontal lobe at junction of its anterior and middle thirds; subcortical, except at outer extremity, where hæmorrhage had broken through the surface; small laceration of anterior fourth of second right temporal convolution, mainly subcortical; cortical hæmorrhage in right middle and posterior fossæ, and to a small amount in right anterior fossa; moderate general hyperæmia, with a few minute coagula.

CASE LXV. *Symptoms.*—Consciousness lost and partially restored; persistent occipital pain; admission to hospital four days later; stupor merging in final unconsciousness; loss of control of bladder and rectum; right radial pulse fuller and stronger than the left, but difference not strongly marked; pupils normal; temperature on admission 100°; normal, with exception of eight hours on the seventh day, when it was 99·2° to 99·4°, till ten hours ante mortem; final temperatures 99·2° to 103·8°; pulse, 45 to 80; respiration, 14 to 18. Death in ten days.

*Lesions.*—Hæmatoma in left occipital region, mainly upon left side; biparietal and left parieto-occipital sutures loosened but not separated; small laceration on under surface of right frontal lobe anteriorly; cortical hæmorrhage covered the whole lateral and superior surfaces of both hemispheres, except in left lower parietal region, extended into median fissure and beneath tentorium over superior surface of cerebellum, and occupied both anterior and both middle fossæ. The effusion was thin, except at the base and over the frontal lobes, where the clot was thick, firm, black, and closely adherent to the cortex, and could be traced into the frontal laceration from which it originated. A still smaller laceration existed upon the inferior surface of the right temporo-sphenoidal lobe. The brain was moderately hyperæmic and very œdematous in its cerebral portion. There were no punctate extravasations, few minute coagula, and no inflammatory products.

Case LXVI. *Symptoms.*—Complete unconsciousness, which continued till death; hæmorrhage from nose and mouth; pupils contracted and immovable, but in a few hours the left became dilated; some convulsive movements of the right arm; retention of urine; second day, ecchymosis of both eyes and subconjunctival hæmorrhage in the right; continued dilatation of the left pupil; right normal; temperature on admission 101°; in four hours, 102°; in sixteen hours, 105°, and in twenty-four hours, 106°; pulse and respiration frequent throughout. Death in twenty-six hours.

*Lesions.*—Extravasation of blood into the substance of left temporal muscle disclosed by incision; open fissure extended from squamous portion of right temporal bone across both orbital plates and intervening cribriform plate of the ethmoid, broke off left lesser wing of the sphenoid, crossed left middle fossa and petrous portion, and terminated in left margin of foramen magnum; epidural clot occupied the whole right anterior fossa, and another of smaller size existed in the left middle fossa; a thrombus filled the posterior part of the superior longitudinal sinus; cortical hæmorrhage over superior surface of the cerebellum, derived from a small laceration of its lateral border; small pial hæmorrhages over left parietal and temporosphenoidal lobes, and a larger one over right parietal lobe; large subcortical laceration of left temporo-sphenoidal lobe, excavating its substance beneath the second, third, and anterior portion of first convolutions, which did not reach the surface; moderate general hyperæmia more marked in the pons and cerebellum.

Case LXVII. *Symptoms.*—Patient walked two miles to the hospital gate and was carried unconscious to the ward; ecchymosis of right eye and wide dilatation of right pupil, slight contraction of the left; no motor or sensory disturbances; left brachial pulsation full and strong, the right very small and weak; same conditions existed in the radial arteries, but the contrast somewhat obscured by contusion of the left wrist; temperature on admission, 98°; four hours later, 104·6°; immediately after death, 105°; half an hour post mortem, 105·4°; pulse, 40 to 64; respiration, 32 to 36; cyanotic just before death, at the end of five hours.

*Lesions.*—Contusion of scalp, disclosed by incision, extending from coronal suture backward above the temporal ridge; fracture in right middle fossa, involving both squamous portion of temporal and greater wing of the sphenoid bone; firm epidural clot from laceration of anterior branch of the arteria meningea media, measuring three fluidounces, which filled the right middle fossa and flattened the temporal lobe laterally and inferiorly. When the clot was removed the brain retained its position, widely separated from the base, and leaving the anterior petrous surface and the adjacent middle fossa exposed. The smaller superficial veins and arterioles of the brain were congested and the surface between them, at first pale, was soon uniformly reddened. There was a small laceration of the posterior part of the third left temporal convolution: another somewhat smaller than a buckshot was found in the anterior part of the pons at the apposition of the longitudinal fibers. The brain substance was generally hyperæmic, especially in the left hemisphere, but without minute extravasations or thrombi. The surfaces of section soon became deeply reddened and bathed in watery effusion.

Case LXVIII. *Symptoms.*—Partial unconsciousness; recurrent hæmorrhage from right ear, succeeded by a flow of serous fluid; vomiting; dilatation of both pupils; retention of urine; greater fullness and strength of left radial pulse than of the right; mental condition normal; intercurrent bronchitis on the third day, which ran its usual course; from the second day a peculiar dusky and swollen appearance of the face, which continued till within two or three days of death; no other indications of cerebral injury till the fourteenth day, when there was occipital pain, which became general headache, and a little later somnolence and occasional irritability. On the eighteenth day, the fifth of this epoch, posterior cervical rigidity; delirium; temperature at its maximum; tenderness along the course of the larger nerves of the left lower extremity from the twentieth to the twenty-fifth days; delirium more active, lucid intervals less frequent, somnolence more continuous, and sense of hearing impaired; deafness progressive till complete power of articulation gradually lost, and finally communication only possible by gesture; dysphagia occurred more suddenly and a little later. The mental condition varied from normal to that of stupor or delirium; emaciation was progressive; paralysis and hyperæsthesia of the left lower extremity were of late occurrence; recurrence of posterior cervical rigidity was once noted, but was transitory; toward the end control of urine and fæces was lost; during the last twelve hours unconsciousness was complete, and respiration rapid, insufficient, and entirely nasal. Death occurred on the thirty-first day. The temperature on admission was 97°, became normal in four hours, and was afterward 99° till the invasion of bronchitis, on the third day, when it rose to 103°, and subsided with recovery from the complication. On the tenth day it again rose with the recurrence of intracranial symptoms to 103·4°, and afterward varied from 100° to 104°, and was not often less than 101°+. The post-mortem temperature receded in half an hour from 103·4° to 103°. The axillary temperatures, carefully recorded from the sixth day, were symmetrical in nearly half the observations, and in the others usually varied two tenths of a degree, and were rather more frequently higher on the right side. The pulse was usually from 64 to 90. The respiration, always frequent, was rarely less than 30 in the minute from the time of admission.

*Lesions.*—No external injury; linear fracture extending from squamous, through petrous portion of right temporal bone; simple thrombosis of lateral sinuses from torcular Herophili into jugular veins; punctate extravasations in pia mater; large occipital veins distended; no serous effusion at the vertex; but patches of false membrane mainly upon left frontal lobe, and upon either side of the median fissure. Several ounces of turbid serous effusion at the base, and a large amount in the lateral ventricles; fibrinous exudation covering the pons, medulla, and inferior surface of the cerebellum, one to two millimetres in thickness, and in the Sylvian fissures; limited contusion of posterior part of the surface of the right temporo-sphenoidal lobe, covering a space an inch square, which was of a yellowish color and studded with hard miliary hæmorrhages; fornix much softened, and brain substance generally hyperæmic and œdematous.

Immediate microscopic examination showed the membranous effusion to be crowded with small round cells which extended for some distance in diminishing quantity into the substance of the underlying cerebellum. Other portions of the brain tissue were unchanged. The *Streptococcus pyogenes* was developed from cultures of the exudation.

Case LXIX. *Symptoms.*—Consciousness lost, but restored at time of admission; hæmatoma over right frontal region; vomiting; severe frontal headache; face flushed; pupils normal; temperature, 95°; pulse, 90; respiration, 20. One hour later, wide dilatation of right pupil, and right cornea more sensitive than the left; sudden unconsciousness, followed by rigidity of the left side, and convulsive movements of the right. At the end of an hour and a half, temperature, 97°; pulse, 85, and Cheyne-Stokes respiration. Death in three hours from time of injury.

*Lesions.*—Linear fracture in squamous portion of right temporal, continued through anterior part of middle fossa, and terminated in the body of sphenoid bone; large epidural hæmorrhage over lateral surface of the right hemisphere nearly to the median fissure; blood partially coagulated and derived from posterior division of the arteria meningea media; surface of the right temporal and anterior part of the right occipital lobe somewhat flattened; slight contusion of left second temporal convolution; brain moderately hyperæmic and œdematous.

Case LXX. *Symptoms.*—Gunshot wound, immediate unconsciousness; rapid and feeble pulse; sighing respiration; profuse hæmorrhage from wounds of entrance and exit. Death within an hour.

*Lesions.*—Gunshot fracture of right frontal bone through temporal fossa; ball grazed the outer and posterior part of the orbital plate and fractured the right lesser wing of the sphenoid, grooved the inferior surface of both frontal lobes just anterior to the fissures of Sylvius, destroying the cortex and subcortex for a space three fourths of an inch in width, and emerged through the left temporal fossa at a little higher level than the point of entrance. The vertex and base were fissured from the point of exit, and the coronal and biparietal sutures divulsed and widely separated. The whole surface of the brain was covered by a thin subarachnoid hæmorrhage which was partly cortical and partly pial. The brain substance generally was markedly hyperæmic and its minute vessels filled with coagula. The corpora striata and optic thalami, especially the striata, were much contused, their substance studded with punctate extravasations, and their vessels distended with thrombi. The pons, medulla, and cerebellum were but slightly altered.

CASE LXXI. *Symptoms.*—Coma; stertor; pulse strong and irregular; respiration slow; slight dilatation of both pupils, which were insensitive; slight twitching of both arms; extremities cold; no external evidence of injury; temperature, 94·2° to 101·2°; respiration, 24, 20, 14; pulse, 42 to 52. Death in eleven hours.

*Lesions.*—Linear fracture extended from just above and behind right ear into the posterior inferior fossa; enormous epidural hæmorrhage, derived from the posterior division of the middle meningeal artery, which compressed the right hemisphere; slight laceration of the right parietal lobe, posterior to the fissure of Rolando.

CASE LXXII. *Symptoms.*—Unconsciousness, which continued till death; contusions and superficial wounds of the left side of the face and temporal region; dilatation of both pupils, of the right more than of the left; hæmorrhage from mouth, nose, and right ear; relaxed muscles, and imperceptible pulse at the wrist. Death occurred five minutes after admission, and in about an hour after reception of injury.

*Lesions.*—Hæmatoma over left temporal, both parietal, and right occipital regions, from rupture of the intracranial vessels; calvarium crushed on the left side anteriorly, and its fragments deeply depressed and distorted; zygoma and both orbital plates comminuted; body of the sphenoid bone disintegrated, and the base of the skull extensively fissured; thin pial hæmorrhage covered the entire brain, possibly augmented by some cortical effusion at the base; limited contusions confined to the cortex about the right parieto-frontal junction and along the right side of the median fissure; cortical lacerations upon the inferior surface of the left frontal lobe and at the tip of the left temporo-sphenoidal lobe; brain substance generally hyperæmic and œdematous, with a few punctate extravasations.

CASE LXXIII. *Symptoms.*—Patient was found in the early morning, sitting in a chair, in which he was said to have passed the night. He would not reply to questions, from seeming lack of comprehension. He could walk, though he had little control over his limbs; his face was pale and showed traces of vomiting. On admission, there was partial consciousness, right hemiplegia and hemianæsthesia; dilatation of left pupil and contraction of the right; loss of control of urine and fæces; profuse serous discharge from both ears, and slight œdema of the lungs. Two hours later coma was complete and œdema of the lungs had increased. Death occurred in ten hours. Temperature on admission, 99·2°; in two hours, 101·2°; in four hours, 103°; in six hours, 103·4°; in nine hours, 103·8°; in ten hours, when *in articulo mortis*, 104°; thirty minutes post mortem, 106°. The right axillary temperature was 0·2° higher than the left at each observation. Pulse, 90 to 108; respiration, 36, 30, 38.

*Lesions.*—Slight hæmatoma over right parietal region disclosed by incision; fracture extending from right of occipital tuber, across petrous portion, into greater wing of sphenoid; opacity of arachnoid in right frontal and anterior parietal regions; small cortical hæmorrhage over frontal lobes; extensive laceration of the frontal, temporal, and inferior portion of parietal lobes on the left side; these parts were excavated and filled with a dark solid clot which was extruded in large quantity through a long tear made in the process of removal of the brain from the cranial cavity; slight ante-mortem cortical rupture through which a little blood had escaped into the middle fossa and ascended upon the frontal region, and another into the posterior cornu of the lateral ventricle, through which the chorioid plexus was infiltrated; small, deep laceration upon the anterior border of the left cerebellum; moderate general hyperæmia and marked œdema, with a few punctate extravasations; thrombi in the superior longitudinal and lateral sinuses.

Case LXXIV. *Symptoms.*—Pistol-shot wound through right temporal fossa; cutaneous opening small and circular; consciousness permanently lost; pupils dilated, the right slightly the larger; left corneal reflex absent; urine retained; some pulmonary œdema; temperature on admission, 96·4°, and from 96·8° to 96° for five hours, then declined to 95·6°; pulse on admission, 85, subsequently from 94 to 80; respiration on admission 14, for an hour and a half 12, in two hours and a half 10, in three hours 8, in four hours and a half 6, in five hours 4, and *in articulo mortis*, a few moments later, 2. Cardiac action continued three minutes after respiration ceased.

*Lesions.*—Ball entered the cranial cavity, severing the trunk of the middle meningeal artery, passed through the third right temporal convolution, and was lodged in the posterior part of the inner border of the right temporo-sphenoidal lobe. A small piece of bone, driven in advance of the ball, was found between the right lateral columns of the medulla. A large arachnoid clot, probably in part epidural and in part cortical, filled the middle fossa, spread over the whole right hemisphere, and thickly covered the pons and medulla. Hyperæmia of the right hemisphere and basic ganglia was of considerable intensity.

Case LXXV. *Symptoms.*—Fell down a flight of stairs; still unconscious on admission; wound in left occipito parietal region; hæmorrhage from left ear; right pupil irresponsive and widely dilated, left pupil moderately dilated; muscular system relaxed; temperature on admission 95° and in an hour normal; rose progressively to 104·2°; right axillary temperature uniformly from 0·2° to 0·4° higher than the left till the last observation, when the difference was 2°; respiration 22 to 24; pulse on admission 72, irregular and intermittent, and afterward 78 to 86 till immediately before death, which occurred in eight hours and a half.

*Lesions.*—Fracture which extended from the left inferior occipital curved line through petrous portion into sella turcica; laceration, two inches long by an inch wide, of the inferior surface of the left temporo-sphenoidal lobe; another, half an inch in diameter, at the anterior extremity of the first left temporal convolution; and a third upon the inferior surface of the right frontal lobe, which involved its anterior half; cortical hæmorrhage filled right anterior and both middle fossæ, covered the right hemisphere laterally, and extended as a thick clot over the right frontal lobe and along the corpus callosum quite to the cerebellum; some small extravasations in the substance of the pons; general hyperæmia and punctate extravasations in the anterior and posterior portions of the brain.

Case LXXVI. *Symptoms.*—Coma; stertor; left pupil dilated; small wound and larger hæmatoma in left parietal region; sensation diminished in both lower extremities and muscular twitching in the right; vomiting; pulse 52. After trephination a soft epidural clot was discovered and a considerable loss of blood ensued. Using as a guide a fissure which extended through the squamous and petrous portions into the middle fossa, the bone was chiseled and the posterior division of the middle meningeal artery, which was found to be the source of hæmorrhage, was clamped. The pulse increased in frequency to 72 to 104, the pupils became normal, but consciousness was not restored, and death occurred a few hours later.

*Lesions* as above.

Case LXXVII. *Symptoms.*—Unconsciousness and death immediately after admission.

*Lesions.*—Skull crushed and flattened on right side; fragments very movable; comminuted on the left side; extensive laceration of the brain posteriorly in the left hemisphere; only small superficial wounds of the scalp.

Case LXXVIII. *Symptoms.*—Coma; stertor; hæmorrhage from left ear; contusion of left parietal region; pupils dilated; pulse full and slow; temperature on admission 98°, and rose progressively to 103·6° at time of death in four hours; no decrease for one hour post mortem; respiration 18 to 26; pulse on admission 70 and rose to 90.

*Lesions.*—Fissure extended from left parietal eminence, through squamous and petrous portions into middle fossa; deep laceration of inferior surface of right temporo-sphenoidal lobe and of lateral border of right cerebellum; cortical hæmorrhage filled right middle fossa; hyperæmia of the right side of brain.

Case LXXIX. *Symptoms.*—Consciousness lost and not regained; coma grew more profound; slight œdema of scalp in right temporal region; pupils slightly dilated; great restlessness and irritability; lack of urinary control; temperature on admission 100·4° and rose to 108°, with only two or three brief fractional recessions; pulse, 94, 58, 80; respiration, 28 to 24. Death in forty-three hours.

*Lesions.*—Hæmatoma over whole right side of the head; linear fracture from right frontal through parietal bone into the inferior occipital fossa; large epidural clot over the whole base on the right side, extending upward over the lateral surface of the brain; laceration of the inferior surface of both temporo-sphenoidal and both occipital lobes; laceration of the inferior surface of both frontal lobes in their anterior portion, very extensive on the left side; cortical hæmorrhage over posterior border of the cerebellum; extensive general hyperæmia with punctate extravasations.

Case LXXX. *Symptoms.*—None; patient found dead.

*Lesions.*—Large lacerated pistol-shot wound in right temporal region; temporal muscles burned and disintegrated for some distance from the cutaneous opening. The ball passed through both frontal lobes, comminuted both orbital and intervening cribriform plates, and emerged through left temporal fossa. The calvarium was separated from the supraorbital ridges and broken into large loose fragments in its anterior portion.

Case LXXXI. *Symptoms.*—Large hæmatoma over left frontal region; epistaxis and hæmatemesis; simple fissure from left frontal eminence into the orbital plate disclosed by incision. On the sixth day, muscular twitching of the whole right side, including the extremities, but not the face, which ceased entirely in fourteen hours and was followed by left hemiplegia and hemianæsthesia. On the seventh day a convulsion, confined for thirty minutes to the right side but afterward becoming general, occurred two hours before death. Temperature on admission was 100·2°, rose to 104·4° on the same day, and to

105·6° on the next, with recessions, and afterward varied from 102° to 105·2°, with no observation for six hours ante mortem. Pulse on admission was 120, and subsequently 130 to 152. Respiration 26 on admission, and later 44 to 58.

*Lesions.*—Fracture extended from the orbit through posterior part of the ethmoid and body and right lesser wing of the sphenoid into the floor of the right middle fossa; general subarachnoid purulent effusion most marked in the left frontal region below the site of fracture.

Case LXXXII. *Symptoms.* — Conscious on admission; Cheyne-Stokes respiration; dilatation of left pupil; right radial pulse fuller and stronger than the left; hæmatoma on the right side of the head anterior to the occipital junction, and small lacerated wounds over both frontal eminences; muscular contractions of left side, and later of both sides of the body. On admission, temperature, 99·3°; pulse, 104, and respiration, 19.

*Lesions.* — Multiple fracture; fissure across frontal bone above the orbits, extending on either side through the parietal bone to the median line of the vertex on both sides, thence to the occiput, and on the right side behind the ear to within an inch of the foramen magnum; another fissure on the left side extended through the orbital plate of the frontal and lesser wing of the sphenoid into the middle fossa. The dura and pia were lacerated from right mastoid region to a point just beyond the median line. The right motor area was extensively lacerated, and the right optic thalamus and corpus striatum to a lesser extent. The left hemisphere was uninjured.

Case LXXXIII. *Symptoms.*—Suicidal gunshot wound; ball entered just below right ear and in front of the mastoid process, and was lodged in the petrous portion of the temporal bone; removed on the following day; delirium and rise of temperature on the sixth day, flexion of the right leg on the thigh on the eighth day, and death on the fourteenth day.

*Lesions.*—Fracture of anterior surface of the right petrous portion, epidural and arachnoid hæmorrhage at that point, pial hæmorrhage over left occipital lobe and left motor area, and laceration of the temporo-sphenoidal lobe at the site of fracture.

CASE LXXXIV. *Symptoms.*—Suicidal wound through the anterior cervical region; ball of 0·38 caliber entered in median line over the larynx; no hæmorrhage from the wound or mouth; immediate partial aphonia; deglutition of liquids only possible and with difficulty; hæmorrhage from left ear. On the third day, deglutition impossible and mental condition stupid; followed by delirium requiring mechanical restraint on the fourth day, and on the fifth day, by paresis of right arm, hand, and lower extremity, and loss of fæcal and urinary control, with some improvement in voice and power of deglutition. On the sixth day there was added right facial paralysis with ptosis; the right pupil was dilated and the left contracted, and the urine was controlled. The temperature on admission was 100°, rose on the second day to 101°, on the third day to 102·4°, and on the fourth day to 103·6°; later it attained an elevation of 107·6°. The pulse was from 70 to 86 till the fourth day, when it rose to 132. The respiration was normal for four days and became frequent only at a late period. Death occurred on the seventeenth day.

*Lesions.*—Bullet was lodged in the apex of the left petrous portion; small fragment of bone driven upward about an eighth of an inch; no lacerations; large pial hæmorrhage, in greatest amount over left fissure of Rolando; large subarachnoid serous effusion.

CASE LXXXV. *Symptoms.*—Primary and permanent unconsciousness; wound above right superciliary ridge; ecchymosis of both eyes; stertor; hæmorrhage from mouth, nose, and both ears; left pupil dilated, the right contracted, and both insensitive, and fibrillar twitching of the right chest muscles. No paralysis or muscular rigidity. The temperature on admission was 99·4°; pulse, 120, full and strong, and the respiration 13; the temperature rose to 99·6°, and the respiration was reduced to 4. Death occurred in twenty minutes; immediate post-mortem decline in temperature.

*Lesions.*—Extensive comminuted fracture of frontal bone and both frontal plates extending through the middle fossæ into the petrous portions; the left optic nerve was crushed by a fragment of bone in the optic foramen. The inferior surface of both frontal lobes was deeply lacerated over its whole extent, and a cortical hæmorrhage, still fluid, occupied all the basic fossæ, and covered the pons and medulla.

CASE LXXXVI. *Symptoms.*—Primary and permanent complete unconsciousness; hæmorrhage from left nostril; dilatation of both pupils; no convulsions or muscular rigidity; respiration not more than four to five in the minute at any time after the receipt of injury and finally not more than one; pulse continued full, strong, and of normal frequency for some moments after respiration ceased. Death in forty-five minutes.

*Lesions.*—Fracture extending through left side of the base into middle fossa; moderate pial hæmorrhage covering whole surface of the brain, vertex, and base, and also the medulla; marked general hyperæmia and œdema; contusion of under surface of left temporo-sphenoidal and frontal lobes.

CASE LXXXVII. *Symptoms.*—Primary and permanent unconsciousness; stertor; dilatation of the pupils; loss of urinary and fæcal control, and pulmonary œdema; left radial pulsation fuller and stronger than the right; no external injury. Temperature, 104° to 104·8°; pulse, 120 to 166; respiration, 24 to 52. Death in four hours and a half.

*Lesions.*—Fracture extending into both occipital fossæ, and a fissure from *contre-coup* in the right middle fossa; large epidural hæmorrhage from *contre-coup* over right frontal region; complete excavation of right frontal lobe with rupture of inferior cortex and consequent cortical hæmorrhage over superior surface of whole right hemisphere and left frontal lobe; contusion of third left temporo-sphenoidal convolution and small extravasation into center of the pons; general hyperæmia.

### FRACTURES CONFINED TO THE VERTEX.

CASE LXXXVIII. *Symptoms.*—Delirium on the second, and a convulsion on the fifteenth, day after the original injury alone noted and significance not recognized. Late symptoms followed an operation for fractured patella with use of anæsthetic six months afterward; general convulsions on the succeeding day, with wild delirium, and temperature 103°; the temperature and general condition became normal after twenty-four hours. One month later general convulsions recurred after another operative interference, and continued thirty-six hours, preceded by tonic spasm of affected (left) limb, and succeeded by delirium and death at the end of nine hours. Each convulsion was preceded by restlessness and wide dilatation of both pupils, and in about fifteen seconds began in the left face, extended to the right face, to the left extremities, and finally became general. Temperature rose in twenty-four hours from 101·1° to 104·8°, and afterward declined to 104°.

*Lesions.*—Extensive laceration of the right temporo-sphenoidal lobe, three inches and a half by an inch and a half in its diameters, involving almost the whole of the second and third, and a little of the first, convolutions; the whole lobe was greatly atrophied, indurated, and pigmented. Circular laceration upon the anterior border of the right frontal, and another, an inch and a half in diameter, upon the inferior surface of the left frontal lobe, in the second and third orbital convolutions. These lacerations were all necrotic.

Case LXXXIX. *Symptoms.* — Hæmatoma of left parietal region; unconsciousness which was permanent; right facial paralysis, and rigidity of both arms and right leg; and twenty-four hours later, paralysis and rigidity of right arm; paralysis of right leg probable. Temperature on admission, 102·6°; pulse, 96; respiration, 36; later temperature, 105°. Death in thirty hours.

*Lesions.*—Epidural hæmorrhage compressing laterally the whole left cerebrum; general hyperæmia and punctate extravasations.

Case XC. *Symptoms.*—Consciousness partially lost and soon regained; vomiting frequent; later, somnolence and coma. Temperature on admission, 100·2°; pulse, 48; temperature rose to 105·4°. Death in twenty-seven hours.

*Lesions.*—Compound comminuted fracture of right frontal bone; corresponding laceration of right frontal lobe, through subcortex nearly to lateral ventricle, with cortical hæmorrhage extending over parietal region; slight pial hæmorrhage over left occipital lobe; minute vessels filled with coagula in all parts of the brain.

Case XCI. *Symptoms.*—Coma; stertor; pulse and respiration slow; second day—right hemiplegia; eyes deviating to the right; pupils normal; pulse feeble and rapid; respiration inadequate from pulmonary œdema. First temperature, some hours after admission, 101·6°; second day, 103·8° to 105·4°; third day, 106·4°. Death in sixty hours.

*Lesions.*—Laceration of left temporo-sphenoidal lobe extending into occipital region, with cortical hæmorrhage over left motor area, and to base of occipital lobe; general hyperæmia and thrombosis.

Case XCII. *Symptoms.*—Unconsciousness and irritability which continued one week. Temperature, 99° to 100°; second and third weeks, delirium and continued irritability; fourth week, apathy, rambling speech, and delusions, after which patient was transferred to another hospital, where he died after operation.

*Lesions.*—Fracture in left occipito-parietal region; laceration of inferior surface of both frontal lobes.

Case XCIII. *Symptoms.*—Coma; stertor; rapid pulse. Temperature fell to 95°. Death in four hours.

*Lesions.*—Gunshot fracture of right frontal bone; ball entered anterior extremity of fissure of Sylvius, traversed right frontal lobe just below the cortex, parallel to its curve and a little backward, crossed median fissure into left parietal lobe, impinged upon the left parietal bone, which it fractured, and fell back into its track half an inch below the surface, where it rested; little intracranial hæmorrhage.

Case XCIV. *Symptoms.*—Coma, soon becoming profound; normal pupils; general muscular twitching. Death in twelve hours.

*Lesions.*—Gunshot fracture of right frontal bone; ball traversed right hemisphere nearly in its antero-posterior diameter, just above corpus callosum, impinged upon inner surface of occipital bone, and fell into inferior occipital fossa above the dura; considerable cortical hæmorrhage.

Case XCV. *Symptoms.*—Gunshot wound of right temporal region; unconsciousness; no other immediate general symptoms; pulse, 70; temperature, 99°; consciousness soon restored; mental processes normal but sluggish; some discharge of brain matter followed an unsuccessful attempt to locate and remove the ball on the second day; wound afterward practically healed; mental condition apathetic, rational, but without any manifestation of interest in surrounding persons, things, or circumstances; urine and fæces voided without any indication of consciousness. Temperature, 100°+ to 103·6°, usually 101°+. Death in thirty days.

*Lesions.*—Gunshot fracture of right frontal bone; ball entered middle of right third frontal convolution, passed through central portion of both frontal lobes to a point just behind ascending arm of fissure of Sylvius in upper portion of island of Reil, and rested in a cavity five eighths by seven eighths of an inch in its diameter. surrounded by clot and brain detritus.

Case XCVI. *Symptoms.*—Shock; consciousness retained; restlessness and delirium; temperature, 100·2°; rose to 104·6°. Death on the third day.

*Lesions.*—Compound fracture of left frontal bone with corresponding laceration of brain and meninges; general hyperæmia and thrombosis.

Case XCVII. *Symptoms.*—Unconsciousness; dilatation of pupils; rapid pulse and respiration; temperature, 100°. Death in four hours.

*Lesions.*—Deep laceration of inferior surface of right temporo-sphenoidal and slight laceration of anterior border of left temporo-sphenoidal lobe; pial hæmorrhage over superior surface of both hemispheres.

Case XCVIII. *Symptoms.* Unconsciousness followed by delirium soon after admission; extensive wounds of the scalp; normal pupils, right becoming dilated a little later; temperature, 98·2°; in two hours, 99°; pulse, 76; respiration, 22; delirium increased; pulse and respiration unchanged. Death in four hours.

*Lesions.*—Compound double camerated fracture, involving right parietal eminence; skull very thick and unsymmetrical; posterior fossæ large, middle and anterior fossæ contracted; extensive pial hæmorrhage, confined to meshes of pia, forming a thin sheet which covered superior and outer surface of right hemisphere and inferior surface of both occipital lobes; very marked general hyperæmia, especially on right side and in pons and medulla; some minute hæmorrhages upon posterior border of right cerebellum and upon the medulla.

Case XCIX. *Symptoms.*—Temporary unconsciousness; no other primary general symptoms; temperature on admission, 98·4°; second day, 101·6°; afterward, 99°+. On the tenth day, restlessness and slight delirium; eleventh day, slight chill and increased delirium, which became permanent, but of less active character; fourteenth day, post-cervical rigidity; and on the fifteenth, slight general convulsion; mental condition sluggish; pupils remained normal; respiration, 18 to 22; pulse, 104 to 112. Temperature on the evening of the tenth day rose to 101° and on the eleventh day to 104·6°; it varied from that point to 103° till the fifteenth day, when it rose progressively and reached 107·4° on the sixteenth day, and death ensued.

*Lesions.*—Compound fracture with slight depression above right supra-orbital ridge, confined to external table; subarachnoid purulent effusion over both frontal lobes, encroaching upon parietal and extending into median fissure.

Case C. *Symptoms.*—Gunshot wound of right side of the head. Left facial paralysis on second day; hernia cerebri on the third day; mental condition deteriorated and paralysis increased. Patient transferred to Bellevue on the thirtieth day; then suffering from hysteria and melancholia which had preceded the infliction of the injury; restlessness; loss of control of urine and fæces; left hemiplegia; slight dilatation of pupils; articulation difficult; sensation normal; pain in right supra-orbital region and at seat of the wound; mental processes slow. Temperature, 100°; pulse, 120 to 140; respiration, 20. At site of injury there was an infected granulating wound through which a probe could be passed into the brain. Four days later, under ether, an attempt was made to locate the ball, and a cavity was found to exist, extending nearly transversely inward two inches and a half, with moderately firm and well-defined wall, and having a small bit of bone at the bottom. The ball was not discovered. Temperature from admission had risen to 102·6° at time of exploration. Death occurred two days later; temperature then 107·4°.

*Lesions.*—Gunshot fracture of right temporal bone in squamous portion; osseous wound had been enlarged by trephination; slight hæmorrhage over right occipital lobe and a few threads of yellow exudate in same region and on the right side of the median fissure; ball passed through lower face area, nearly transversely inward to a point beneath the median surface and just above the calloso-marginal fissure; was then deflected backward at a right angle by the resistance of the falx cerebri, and was lodged an inch behind the cavity recognized at the time of exploration. General hyperæmia.

Case CI. *Symptoms.*—Gunshot wound of left side of the head. Unconsciousness, which continued till death, five hours later; slight dilatation of left pupil. Temperature one hour after reception of injury 98·2°; two hours afterward, 97·6°; fifteen minutes before death, 99°. Pulse, 118 to 132; respiration, 28; later, 32 and stertorous; fifteen minutes before death, 7; and finally 2.

*Lesions.*—Gunshot fracture of squamous portion of left temporal bone in its posterior portion, an inch below temporal ridge; foyer of entrance triangular; each arm half an inch in length; bone comminuted, and the fragments penetrated the cerebral cortex. Ball entered temporal lobe between two large branches of the meningeal artery, passed transversely across the brain immediately below the cortex, and was lodged in the right parietal lobe; cortical hæmorrhage from injury of the right parietal lobe by the ball in its course, extended under the tentorium and over the pons and medulla, and was apparently the immediate cause of death; cerebral hyperæmia confined to the vicinage of the bullet track.

Case CII. *Symptoms.*—Unconsciousness, which continued till death at the end of three hours; general muscular rigidity. Temperature, 101°; pulse, 98; respiration, 20.

*Lesions.*—Penetrating wound and fracture of left temporal bone, above the ear, three eighths of an inch in diameter, from a blow inflicted with a revolving screw-driver. The instrument passed through both hemispheres, wounding the dura upon the opposite side, and involving the posterior part of the left corpus striatum and both optic thalami; a thin cortical hæmorrhage covered both hemispheres and the superior surface of the cerebellum.

Case CIII. *Symptoms.*—Patient, nine days previous to admission, came home with head bleeding, vertigo, nausea, and feeling of weakness, from an injury of unknown origin, and was said to have been afterward treated for pneumonia. On admission, he was found to have compound depressed fracture of right parietal bone, and the wound was foul and suppurating; mental condition stupid; left hemiplegia and right facial paralysis; deviation of tongue to the left; opposite radial pulsations symmetrical; slight dilatation of left pupil; coma supervened an hour later, and convulsive movements of the right face four hours and a half after admission. After elevation of the depressed bone, and escape of a small amount of pus from below the dura, the pupils became normal, and there was a single clonic convulsion of the left side. Death occurred thirteen hours and a half from time of admission. Temperature for twelve hours was 106°+, and afterward 107·2°; one hour post mortem, 107·4°. Pulse, 118, 170, 158; respiration, 44 to 60.

*Lesions.*—Compound depressed fracture of right parietal bone, just behind coronal suture, and half an inch from median line; purulent subarachnoid effusion over convex surface of right hemisphere, which anteriorly extended to the base; superficial laceration of right parietal lobe beneath the site of fracture, which was prolonged subcortically, both anteriorly and posteriorly, but did not reach the motor area; pus from this laceration had escaped in small quantity into the arachnoid cavity; left hemisphere markedly hyperæmic and moderately œdematous.

Case CIV. *Symptoms.*—Consciousness primarily retained; thirty minutes later general convulsions followed by complete unconsciousness and an apparently moribund condition. Elevation of a depressed portion of the left parietal bone restored consciousness and some strength to the circulation. Convulsions recurred next day, and death ensued in twenty-three hours. Temperature, 102·4° to 104·4°; pulse, 108 to 160; respiration, 32 to 60.

*Lesions.*—Fracture confined to the vertex; epidural hæmorrhage of small extent, and laceration of inferior surface of right frontal and temporo-sphenoidal lobes. (Infant, aged twenty-two months.)

### ENCEPHALIC INJURIES WITHOUT FRACTURE.

CASE CV. *Symptoms.*—Violent delirium for two days; recurred on the sixth day, followed by unconsciousness and hyperæsthesia. Temperature, 103° to 104°; afterward, 100° to 103°; final temperature, 103°. Death in twelve days.

*Lesions.*—Pial hæmorrhage over left occipital lobe, extending into median fissure; subarachnoid serous effusion.

CASE CVI. *Symptoms.*—None recognized till fourth day, when there were four unilateral convulsions. A single one occurred on the fifth day, and they then continued with increasing frequency till death on the eighth day. Each one began by a twitching of the facial muscles, with head and eyes turned to the left, and extended to the left arm, and finally to the left hand. Temperature on admission, 100°; twelve hours later, 103°; then 103° to 104°, till sixteen hours before death, when it rose to 105°.

*Lesions.*—Extensive laceration of right temporo-sphenoidal lobe, with cortical hæmorrhage over whole right hemisphere.

CASE CVII. *Symptoms.*—Mental condition clear, but dazed, on admission ten hours after reception of the injury; extreme muscular tremor, followed in two hours by a general convulsion; from this time periods of general convulsions, with intervals of unconsciousness or delirium, lasting about six hours, alternated with periods of quiescence of equal length; no initial symptom. Death in two days.

*Lesions.*—Deep laceration of right frontal lobe, anteriorly and externally, extending into parietal region; cortical hæmorrhage, covering right frontal lobe, right parietal lobe anterior to the Rolandic fissure, and the temporo-sphenoidal lobe, both laterally and inferiorly.

CASE CVIII. *Symptoms.*—None. Found dead in an upright position, leaning against a fence.

*Lesions.*—Lacerations and contusions covering greater part of left frontal and temporo-sphenoidal lobes; cortical hæmorrhage over the whole left hemisphere.

Case CIX. *Symptoms.*—Coma; stertor; contraction of pupils; full pulse; rapid respiration. Temperature, 101°+. On the third day coma more profound; dysphagia; continued irritability and restlessness. Temperature, 104·5°. Death in four days; temperature, 107·4°.

*Lesions.*—Small laceration at left parieto-occipital junction; cortical hæmorrhage over posterior part of left parietal lobe; general hyperæmia.

Case CX. *Symptoms.*—Coma, restlessness, and general hyperæsthesia; temperature, 103·4°; pneumonia discovered on the second day. Death on the third day.

*Lesions.*—General hyperæmia, with some punctate extravasations; organized membranous effusion, studded with calcareous nodules, over left hemisphere.

Case CXI. *Symptoms.*—Sudden coma; stertor; double facial paralysis; complete right hemiplegia and hemianæsthesia; temperature, 99° to 103°. Trephination and drainage of serous effusion from the base by position of the head was followed within six hours by return of consciousness, mental clearness, power of articulation, and decline of temperature to 98·6°, and this improvement in condition continued fourteen hours; slight chill then preceded a progressive rise of temperature to 104·6°, and death occurred ten hours later.

*Lesions.*—Interior of left occipital lobe disintegrated by apoplectic clot, which extended into both lateral ventricles; consequent fall from a cab caused a laceration of external border of right cerebellum and cortical hæmorrhage, which spread over the pons into the transverse fissure.

Case CXII. *Symptoms.*—No primary general symptoms; temperature, 100°. Second day, delirium. Fourth and fifth days, headache. Sixth day, restlessness, irritability, and failing strength; mind clear. Eighth day, general muscular rigidity most marked in right side and arm, and, a few hours previous to death, perforating ulcer of the cornea. Temperature, second day, 103·2°; third day, 101° to 100·8°; fourth and fifth days, 103·4° to 103°; sixth day, 106·4°; seventh and eighth days, 105° to 105·2°.

*Lesions.*—General hyperæmia; minute thromboses and moderate œdema, markedly involving basic ganglia and cerebellum, and most pronounced on the left side; thrombi filled both lateral and both inferior petrosal sinuses, and extended into right jugular vein, and were decolorized only near the torcular Herophili.

Case CXIII. *Symptoms.*—Delirium; normal pupils and respiration; temperature, 101·4°; pulse, 114. Later, great hypersensitiveness and irritability. The delirium continued, though it did not prevent rational reply to questions; temperature rose to 103·2° on the fifth day, and afterward fell very gradually to 100°; on the fourteenth day it was 103·4°; and on the fifteenth, five hours ante mortem, it was 103·8°, and one hour post mortem it was 104·2°.

*Lesions.*—Cortical hæmorrhage over both hemispheres and in largest quantity over parieto-occipital junctions; some subarachnoid serous effusion in left frontal region; general hyperæmia with punctate hæmorrhages, most marked on the left side.

Case CXIV. *Symptoms.*—Primary unconsciousness; on admission, forty-eight hours later, muttering stupor; rigidity of left arm; incomplete right hemiplegia, more marked in upper extremities; pulse, 60; temperature, 101°; third day, increased rigidity of left arm; complete hemiplegia; profound coma; pulse, 128; temperature, 105°. Trephination was followed by increased freedom of movement and by some power of articulation. Death on the fourth day.

*Lesions.*—Moderate subarachnoid serous effusion over anterior two thirds of right hemisphere; laceration of left temporo-sphenoidal lobe, excavating and destroying its whole structure; cortical hæmorrhage extending around the circle of Willis and upward upon the occipital lobe, and in patches upon the frontal and parietal lobes.

Case CXV. *Symptoms.*—No external evidence of injury; coma; stertor; rigidity of right side; pulse, 120; temperature, 100°. Death on third day; temperature, 103·2°.

*Lesions.*—Large subarachnoid serous effusion; recent clot in substance of left cerebellum. An old laceration existed upon antero superior surface of left occipital lobe and another upon its inferior surface.

CASE CXVI. *Symptoms.*—Consciousness lost and partially restored before admission, twenty-four hours later; mental condition rational, but comprehension slow; slight dilatation of left pupil. Temperature, 99°, followed by some left paresis and by some dysphagia referred to the left side of the throat. The patient from the time of injury often fell out of bed, always on the right side. Subsequently transient facial paralysis occurred; amount of paresis and of dilatation of left pupil varied from day to day; mental condition deteriorated. Temperature for ten days was 99° + ; later, 100° + to 101°; pulse and respiration nearly normal. Trephination on the fifteenth day discovered a small subcortical cavity in the right leg area containing less than a drachm of yellowish fluid, afterward found to contain leucocytes. The temperature was 99° + till eleventh day after operation, when it rose to 104°; next day, 101° to 104°. Death from asthenia on the twenty-eighth day after admission.

*Lesions.*—Large subarachnoid serous effusion compressing frontal lobes; general hyperæmia with minute coagula. The brain substance around the small subcortical cavity opened during life was softened and contained punctate extravasations.

CASE CXVII. *Symptoms.*—Unconsciousness, which still continued upon admission on the second day; slight dilatation of the pupils; complete left hemiplegia and hemianæsthesia; slight left facial paralysis. Temperature, 106°; pulse, 140; respiration, 30; general convulsions beginning soon after admission, and frequently repeated; initial symptom in mouth and lower face. Trephination same day by house surgeon with negative result. Temperature two hours later, 107·4°. Death in a convulsion five hours after operation. Temperature, forty-five minutes post mortem, 109·4°.

*Lesions.*—General hyperæmia of the brain and membranes; tumor of the size of a pea resting in a small cavity in the left frontal lobe formed by disintegration of surrounding brain tissue.

CASE CXVIII. *Symptoms.*—Condition alcoholic and habit epileptic; fell in an epileptic convulsion; large hæmatoma over left frontal and parietal region; three convulsions within first six hours, the last followed by partial paralysis of left lower face. The temperature on the first day was 101·8°, 102·8°, 100°; second to sixth days inclusive, 100·6° to 102°+; seventh to ninth day, normal; and then for ten days subnormal during the greater part of each twenty-four hours. On the thirteenth day a severe chill was followed by temporary rise of temperature to 101°+; and on the nineteenth day a slighter chill by an elevation of temperature, which progressively increased till death, on the twenty-first day. Until the occurrence of the second chill there were few general symptoms; some remaining paresis and anæsthesia of the right face, more or less mental aberration, and some delusions. After the second chill strength diminished, the mental condition became sluggish, the respiration rapid, and temperature rose to 105·5°.

*Lesions.*—Subcortical laceration and excavation of left prefrontal lobe, with a prolongation backward to a point opposite to the middle of the corpus striatum; no hæmorrhages; large subarachnoid serous effusion and opacity of the arachnoid over the whole vertex; general hyperæmia and œdema.

Case CXIX. *Symptoms.*—Consciousness retained; wound in right parietal region; condition alcoholic; heavy sleep during the first night after admission; afterward constant restlessness; some pain in the back of the head; vomiting of everything taken into the stomach; temperature on admission, 102·6°; second day, 105°; and at time of death, which occurred somewhat suddenly at the end of the third day, 103·8°; pulse moderately accelerated, varying from 120 to 88; pupils and respiration normal.

*Lesions.*—Subarachnoid purulent effusion over both frontal lobes, mainly on the left side, with some general œdema of the pia; scanty fibrinous exudation at the base; and fibrinous patches on inner surface of the dura at the convexity.

Case CXX. *Symptoms.*—Absolute unconsciousness till death, an hour and a half after reception of the injury; small wound behind the right ear; dilatation and immobility of both pupils; respiration on admission, 42; an hour later, 21; ceased at death rather suddenly; no cyanosis; pulse feeble and soon became imperceptible; temperature on admission, 98·6°; an hour later, 98·2°.

*Lesions.*—Probably caused by *contrecoup*, force having been transmitted through the feet and lower extremities; fractures of both tarsi, comminution of both calces and right astragalus, fracture of left leg, and contusion of soles of both feet; pial hæmorrhage to extent of several ounces of fluid blood, mainly at the vertex and in larger part on the left side, extending into median fissure, and which had broken through into the arachnoid cavity; also in considerable quantity upon the inferior surface of the cerebellum, about the median line, and covering the pons; no lacerations; excessive general hyperæmia, most strongly marked on the left side and in the pons, optic thalami, and corpora striata, in the order named; thrombosis of minute vessels generally, but most pronounced in the optic thalami and pons; œdema of the pons.

CASE CXXI. *Symptoms.*—Primary unconsciousness; and on admission mind confused and speech disconnected; four general convulsions from twelve to twenty-four hours afterward; no control of urine or fæces; second day, semi-consciousness; muscular rigidity in back of the neck and extremities; some irritability; fourth day, mental condition rational, but no remembrance of the manner in which the injury had been received; during the next ten days the urine, but not the fæces, remained uncontrolled; there was noticeable weakness of the muscles of the trunk, inability to rise or sit up in bed without assistance, dementia and loss of memory, primary union of the wound, and nearly normal pulse and respiration. On the fifteenth day there was somnolence and increase in temperature and infrequency of the pulse and respiration; stupor deepened, and on the seventeenth day unconsciousness was complete. Death occurred in eighteen days. Temperature on admission, 99·4°; fourth day, 99°; till the end of second week, 99° to 100°+; on the seventeenth day, 102·7° to 103·8°; on the eighteenth day, 105·4°. Pulse on admission, 96; normal till fifteenth day; later, 160. Respiration on admission, 26.

*Lesions.*—Hæmatoma over right parietal eminence; thrombus in superior longitudinal sinus; great fullness of meningeal veins over the vertex; convolutions flattened; frontal lobes relatively small, parietal lobes bulging as though from distention; general cerebral hyperæmia and œdema without punctate extravasations and with few minute thrombi; substance of cerebellum nearly normal. By compressing posterior portion of the cerebrum and making vertical sections anteriorly, serous fluid exuded in great quantity; little serum in the ventricles. A clot about the size of a large pea and of elliptical form occupied the exact center of the anterior third of the left optic thalamus. There were no lacerations, hæmorrhages, or sub-arachnoid effusions, and upon microscopical examination no inflammatory changes.

CASE CXXII. *Symptoms.*—Walking case; unconsciousness supervened some hours after injury, and continued till death on the third day; wounds in occipital and both parietal regions; slight dilatation of left pupil. Temperature, 103·6° to 106·6°.

*Lesions.*—Large pial hæmorrhage compressing left fronto-parietal region; excessive general hyperæmia with numerous minute thromboses; subcortical laceration just external to anterior part of left corpus striatum, an inch by half an inch in its diameters.

CASE CXXIII. *Symptoms.*—None recognized till admission three days after reception of the injury; partial loss of consciousness; complete right hemiplegia and hemianæsthesia including trunk; complete aphonia; slight dilatation of pupils; bilateral convulsive movements of face and neck with the eyes turned to the right, repeated every five minutes; respiration shallow and hurried; pulse rapid, feeble, and irregular. Temperature, 101° to 104°; radial pulsation fuller and stronger on the left side than on the right. Trephination disclosed arachnoid clot. Death occurred before operation was completed.

*Lesions.*—Pial hæmorrhage with clot covering both frontal and both parietal lobes; right lateral ventricle filled with hæmorrhagic serous effusion; general hyperæmia.

CASE CXXIV. *Symptoms.*—Walking case; unconsciousness supervened some hours after apparently trivial injury; no discoverable external lesion; dilatation of pupils; second day, partial restoration of consciousness; fourth day, delusions; ninth day, stupor; eleventh day, complete unconsciousness. Death at end of twelfth day. Temperature on the first day, 102·4°; afterward, 101° to 99°; final observation, 100·8°; pulse, 76, gradually increasing in frequency; respiration, 24, 20, 28.

*Lesions.*—Thin layer of pial hæmorrhage which covered the opposing surfaces of the superior median fissure, and spread over left occipital and parietal lobes to margin of the temporal lobe; some blood, also pial, in the left middle fossa; general hyperæmia and moderate œdema.

Case CXXV. *Symptoms.*—Unconsciousness which soon after admission was replaced by delirium; no external injury; loss of urinary control; delirium constant, of a quiet sort by day and violent by night till the seventh day, when for some hours before death it was muttering, or typhoid, in character; mental condition stupid from the beginning; patient was at no time able to give any account of himself, to respond to a question, or to show any appreciation of his surroundings. Death from asthenia on the seventh day. Temperature on admission, 96·2°; rose progressively in three days to 103·2°; on the fourth day was 101·8°; on the fifth day, 103°; on the sixth day, 104·6°; and on the seventh day, 101·2° to 107·2°; post mortem, 107·8°. The pulse did not exceed 100 till late in the week.

*Lesions.*—Small laceration in the substance of the posterior part of the left frontal lobe; laceration of under part of the corpus callosum in its anterior third, and of left lateral edge of the fornix anteriorly; small hæmorrhage in left lateral ventricle derived from the laceration of the fornix; pial hæmorrhage over posterior part of right occipital lobe, upon its border, beneath tentorium, and upon the posterior border of the cerebellum; blood fluid and moderate in amount; moderate general hyperæmia with minute thromboses.

Case CXXVI. *Symptoms.*—Walking case; unconsciousness after some hours' interval; stertor; loss of urinary control; vomiting. Temperature, 101·8°; rose progressively to 107 8°; pulse, 70 to 162; respiration, 24 to 46. Death in eleven hours.

*Lesions.*—Laceration of superior surface of right parietal lobe; cortical hæmorrhage covering whole right hemisphere; general hyperæmia.

Case CXXVII. *Symptoms.* — Unconsciousness, which soon became profound; normal pupils; pulse in a few moments rose from 90 to 140; right side of body and right extremities rigid; bilateral convulsive movements; right radial pulse fuller and stronger than the left. Death in eight hours and a half. Temperature on admission, 97°; in three hours, 101°; in six hours, 102·2°; pulse, 90 to 140 to 136; respiration, 20, 18, 21; and just before death, 12 and then 7 in the minute, very full and deep, with cyanosis.

*Lesions.*—Small contusion of scalp in left middle parietal region discovered only after post-mortem incision; thin pial hæmorrhage, mostly fluid, covered whole superior and external surfaces of both hemispheres as far forward as the middle of the frontal lobes, extended in larger quantity over both surfaces and both borders of the cerebellum, and spread over the pons and medulla; pia mater intensely hyperæmic; small contusion on inner border of right temporo-sphenoidal lobe, and a larger one at left parieto-occipital junction; brain substance generally excessively hyperæmic and œdematous, with many small areas of local contusion filled with small hæmorrhages as large as a robin shot.

The essential lesion was laceration of the basic ganglia. The right corpus striatum was entirely disintegrated and destroyed; its ventricular surface only remained, as a ragged membranous capsule, of which much had altogether disappeared. The laceration extended antero-laterally into the substance of the right frontal and parietal lobes; it was continued posteriorly through the tænia semicircularis into the anterior part of the optic thalamus. The ventricular surface of the left corpus striatum was contused and marked by small linear lacerations. The fornix and under surface of the corpus callosum were softened and disintegrated. Fluid blood partially filled both lateral ventricles, and in the left had broken through the posterior cornu into the occipital lobe in considerable quantity.

Case CXXVIII. *Symptoms.*—Immediate unconsciousness with some response to external irritations, which continued till final coma; continued dilatation of both pupils, which were sensitive; temporary rigidity of left side; right hemiplegia and hemianæsthesia, and right facial paralysis; restlessness, which was confined to the left side; retention of urine; coma and stertor for five hours before death, which occurred in fifty-three hours. Six hours before death the left hand became icy cold and the left arm and foot cool, while other parts of the body retained a normal surface temperature. At this time the rectal temperature was 102·6°; the left axillary, 100·4°; and the right axillary, 103·2°. In fifteen minutes the temperature in the left axilla rose to 101·4°, and in thirty minutes to 102·8°, while the rectal and right axillary temperatures remained stationary. The axillary temperatures were at other times symmetrical. Temperature on admission was 98·5°, and in two hours, 102·2°; in eleven hours it receded to 101°, in the next twelve hours rose to 105°, on the second day receded to 100·4°, and a few moments before death was 106°; one hour post mortem, 106·2°. The pulse gradually increased in frequency from 110 to 158. The respiration was never below 30, and was finally 56 in the minute.

*Lesions.*—Contused wound of the scalp over right parietal eminence; slight pial hæmorrhage over inferior surface of cerebellum and posterior left occipital border; copious subarachnoid effusion and arachnoid opacity in posterior parietal regions most marked on the left side; small hæmorrhagic serous effusion in left lateral ventricle; limited contusion and slight laceration in the substance of the fornix posteriorly; excessive general hyperæmia and œdema, with a few minute thrombi in all parts of the brain.

Case CXXIX. *Symptoms.*—The patient walked home after a fall of ten feet, had a single convulsion a few hours later, and was stupid or dazed for five days afterward; he then became violently delirious, and was admitted to the hospital. At that time, no visible external injury; pupils moderately dilated; radial pulsations bilaterally symmetrical; posterior cervical muscular rigidity, and loss of urinary control. On the following (seventh) day pupils contracted and muscular rigidity increased; one convulsion after admission; mental condition marked by alternations of stupor, with wild delirium. No change till the eleventh day, when the patient became quieter, and could answer a limited number of questions intelligently. On the fifteenth day the pupils became normal, muscular rigidity diminished, and urinary control was temporarily regained. From the sixteenth day unconsciousness was complete. On the seventeenth day the pupils were again contracted, the respiration was stertorous, and the face cyanotic; the lungs became œdematous, and death occurred on the morning of the nineteenth day. The temperature on admission was 102°, and varied from 99° to 101°+, with occasional elevations to 102°+ till the last thirty-six hours, when it was constant at 105·6°; and half an hour post mortem was 106°. The pulse on admission was 132, and afterward was usually from 96 to 112. The respiration was moderately increased in frequency. Both pulse and respiration were finally greatly accelerated.

*Lesions.*—Cortical hæmorrhage, compressing outer and anterior aspect of right frontal lobe, and filling right anterior fossa. This was derived from a laceration of the inferior surface of the right frontal lobe, mainly subcortical, which excavated its inferior and outer portion; cavity as large as a pigeon's egg and lined by a thin, chocolate-colored and pultaceous substance. Small linear laceration upon inner border of left frontal lobe and slight contusion of anterior portion of right temporo-sphenoidal lobe, both upon inferior surface. Opacity of arachnoid membrane; no subarachnoid serous effusion, and only very moderate hyperæmia of the brain substance.

Case CXXX. *Symptoms.*—Primary and permanent unconsciousness; restlessness; general muscular rigidity; stertor; irregular pupils. Temperature on admission, 100°, and at death 99·8°; pulse varied from 108 to 160; respiration, 32 to 58. Death in an hour and a half.

*Lesions.*—No fracture or lacerations; large general subarachnoid and ventricular serous effusion; general hyperæmia and excessive œdema.

### GENERAL DIAGNOSIS.

Before attempting to isolate the several forms of encephalic injury, I shall recur to two points in their general diagnosis which have been, at least provisionally, established by the analysis of my first series of cases. I refer to the pathognomonic value of temperature in the symptomatology of head injuries as a class, and to the recognition of cranial fracture. I shall not enter at length into the reconsideration of either subject, since the relation between symptoms and demonstrated lesions previously determined is found to still subsist, and the more recent observations have but confirmed the opinions the study of the elder series seemed to warrant. The additional cases have in fact not only substantiated but strengthened the propositions which I formulated in the original instance; this will be sufficiently evident by a simple reference to their histories as presented. I shall consider more specifically only the two points in general diagnosis which I have designated.

### PRIMARY SUBNORMAL TEMPERATURE.

In all the cases of the first series but two the temperature was elevated at the time of first observation. In the present series, which, like the first, includes recovering as well as fatal cases, the instances of primary subnormal temperature have chanced to be more numerous, but in all which survived primary shock subsequent elevation of temperature was equally noted.

In more than seventy-five per cent. of the forty or more cases of the later series in which the earliest known temperature was subnormal, the patient still suffered from evident shock or alcoholic intoxication. It is not inconceivable or improbable that in them a characteristic symptom of shock or of alcoholic poison should have taken precedence of others produced by traumatism. In the cases remaining, where no other suggestion of shock or of alcoholism is to be derived from the examination of symptoms, the solution of the problem is probably the same. Premising that the earliest manifestations of injury—those exhibited before admission—are most difficult to learn, it may well happen that depression of temperature, often the last lingering indication of shock, is all that remains when the first record of the case comes to be made. Three of the residual cases were complicated by severe external injury; in many others there was free intracranial hæmorrhage, as determined in the majority by necropsic examination, and in the others by the escape of blood during life from ear, nose, or mouth. In one case, in which there was no external hæmorrhage, the general conditions indicated its existence within the cranial cavity. In all these instances the occurrence of undiscovered or unnoted shock is not only the rational but more than probable explanation of an otherwise inexplicable early depression of temperature; but whatever may be thought of these exceptional and temporary early conditions, all such cases very soon fall into line and conform to what seems to be an established law—that elevation of temperature is the unfailing manifestation of traumatic lesions within the cranium.

The character of the pulse and respiration in the exceptional instances in which subnormal temperature was observed has not been usually suggestive of general shock; in a few cases the one was notably frequent or the other markedly accelerated; in general there was little if any variation from the normal standard. This requires for explanation but another application of the suggested law of precedence in symptomatology; it may happen that the pulse and respiration reflect for a time the general condition of shock, but it is more frequently the temperature alone which in the presence of intracranial injury is dominated by the original impression made upon the sympathetic nervous system. In all cases, if the patient survives a certain very limited period, temperature, pulse, and respiration, like all other symptoms, are dependent upon the special injury which has been suffered, and this is ordinarily the fact at the earliest opportunity afforded for examination. It is sufficient to indicate this law of preference without attempting to fathom the conditions upon which it rests.

I have previously insisted in this relation of tempera ture to general diagnosis upon the importance of distinguishing alcoholic coma from cerebral trauma. I have no less strenuously asserted the facility, the almost absolute certainty, with which this can be accomplished. I believe the elevation of temperature in the one, and its depression in the other, have been so thoroughly established as to demonstrate the sufficiency of temperature alone, without the existence of external injury or positive general symptoms, in almost any case in which question may arise. It is fortunate that the occasional early depressions of temperature in intracranial injury are likely to be associated with such general conditions as have little room for doubt in diagnosis. I have no reason to revert to a subject so simple in itself, except in the interest of humanity and in protest of the shocking abuses which still persist in this regard in the accident service of the city. Negligence or incompetence still figures in the early history of too many of the serious cranial and intracranial injuries which have at a later period come under my observation. There may be a fair presumption that a man found unconscious in the street, or delirious in a police station, is simply drunk and devoid of surgical interest, but it is not so absolutely overwhelming as to warrant neglect of ordinary physical examination; and when a patient with fractured skull and lacerated brain, whether or not in alcoholic condition, has been given admission, it is not creditable to hospital administration that he should be detained in alcoholic wards, transferred to an asylum for the insane, or sent into the street to die almost within the shadow of its walls, even in exceptional instances. It must be in some part due to a defect in professional teaching when hospital assistants display such ignorance or indifference in the discharge of professional duty. It is well, therefore, for observers who are not public teachers to assume an office, and to direct attention to such default if for nobetter purpose than to avert public scandal.

### FRACTURES OF THE CRANIUM.

The diagnosis of fractured skull is not difficult if the case be subjected to sufficiently careful examination. This is evident in some degree from anatomical considerations, and is illustrated in my first series of cases. Fractures of the vertex can always be discovered by tactile or visual sense, since incision is without danger or subsequent inconvenience to the patient, when doubt exists which it seems important to resolve. Fractures of the base in a large proportion of cases traverse some part of the bone which permits the escape of blood from the ear, nose, or mouth, or into the subconjunctival or subcutaneous cellular tissue. In fifty per cent. of the eighty-seven necropsies in both series in which fracture of the base existed there had been some form of external hæmorrhage during life; in more than seventy five per cent. of the seventy-eight cases which recovered, or in which necropsy was impracticable, there had also been some characteristic hæmorrhage, so that in 62·5 per cent. of the total number diagnosis could be made largely from this single symptom. Two of the remaining sixty cases presented an equally characteristic serous discharge, and many others had been recognized by tracing fissures from the vertex downward into the base in the course of operation. There are left scarcely more than twenty cases in which this fracture was unknown till disclosed at necropsy, and of these, several were brought under observation only after the lapse of one or more days—too late to ascertain whether or not hæmorrhage, usually a transient symptom, had occurred. In this residuum of cases the fracture very frequently extended into the middle or posterior fossa without reaching the petrous portion, and sometimes into that bone without involving any part of the auditory passages; fracture through the anterior or middle fossa in some instances failed to so implicate the ethmoid or sphenoid as to establish communication with the nose or mouth; in the anterior fossa the thin orbital bones were occasionally fissured without causing either orbital or ocular hæmorrhage, visible or concealed. Yet, with all these possibilities of failure of recognition as a symptom, its absence altogether, its lack of means of exit, the neglect of its early observance, external hæmorrhage was noted in nearly four out of five of the whole large number of cases which I have recorded.

I do not think there is the serious difficulty which has been suggested in determining whether such hæmorrhage is the result of fracture. The local examination of ear, nose, or mouth is sufficient to eliminate the most probable source of error. Contusions of the face in the ophthalmic region may sometimes make orbital or ocular hæmorrhage of doubtful significance, and habitual epistaxis has once led me to hesitate in the interpretation of a nasal hæmorrhage; but it is usually possible in such instances to give this symptom its proper clinical value. I believe it may be regarded as practically pathognomonic. I have only once found a hæmorrhage from the ear to result from a wound of the external meatus.

An occasional escape of brain substance through a cranial fracture requires no consideration. The more frequent instance of injury of a cranial nerve from a fracture passing through its bony canal may be diagnostic if it be practicable to fairly determine that functional disturbance or abeyance does not depend upon lesion within the intracranial cavity. I have recorded in the first series an instance of facial paralysis which was found upon necropsy to have been occasioned by fracture and hæmorrhage into the aquæductus Fallopii, and I have had reason in more recent recovering cases to refer the same symptom to similar osseous lesion. It is well known that fracture through an anterior fossa often involves the optic foramen. I have recorded the history of three cases, in which the patient survived, where the optic nerve was thus implicated and suffered subsequent atrophy with immediate and permanent loss of vision. Dr. P. A. Callan has reported nine cases. I have no doubt, therefore, that valuable diagnostic information may be afforded by nerve disturbance mechanically produced.

There remains a symptom which I believe to point to fracture, and to which I have previously adverted, in the existence of acute localized pain at the seat of injury. I have since observed it in a number of cases in which this lesion seemed otherwise probable; these often resulted in recovery, and, as the indications of intracranial complication were slight, the symptom was unobscured. I quote an illustrative case which occurred in my service at St. Vincent's Hospital:

A young woman fell from a third-story window to the pavement below and was admitted at once, delirious, with hæmatoma of the left frontal region extending over the eye, and with slight subconjunctival hæmorrhage. She had epistaxis, which was repeated the next day, and from which she said she had previously suffered; no fracture was discovered by incision; severe frontal headache, confined to the site of the hæmatoma, continued for three days; the mind was clear; the wound of incision healed at once; the temperature on admission was 101·4°, rose gradually to 103° on the third day, and then declined to 99° on the ninth day; the pulse on admission was 68, and on the sixth day 120.

The degree and course of temperature in this case indicated injury of the brain, and while neither the epistaxis nor the ocular hæmorrhage could be positively attributed to fracture, the severity of the blow, amount of local injury, and coexistence of brain lesion, gave to my mind a certain diagnostic importance to a severe localized pain which was certainly not characteristic of simple contusion. I shall have occasion to detail in another connection some fatal cases in which the value of this symptom was incidentally verified.

The symptoms usually ascribed to fracture—as loss of consciousness, pupillary change, and others—are really those of encephalic complication, and have only a possible indirect relation to cranial injury.

In assuming that fractures are themselves unimportant, except for their complications, immediate or remote, I have not depreciated the importance of their diagnosis. Fractures of the vault induce complications, which are relieved only after recognition and treatment of the fracture itself. Laceration of the brain, wounds of the sinuses, hæmorrhages, and later psychical disorders caused by fragments of bone depressed, can be treated only after detection of the primary lesion. Basic fractures are less likely to require or admit direct interference; but their appreciation is still of moment. The knowledge that fracture exists may greatly help to confirm the diagnosis of a deeper seated injury, and greater certainty in regard to the existence and nature of morbid conditions can not fail to increase the possibilities of successful treatment. The curative management of intracranial lesions is still so far unsettled that aid from any quarter, in giving it firmer basis, is far from unimportant.

### DIFFERENTIAL DIAGNOSIS.

When the transition is made from general to special diagnosis, and beyond the simple recognition of fracture to the differentiation of intracranial injuries, difficulties increase, and these, I have found, are to be encountered and surmounted, if at all, with little aid from other than personal observations. The literature of the subject, aside from the contributions of Prescott Hewitt and von Bergmann, is singularly unsatisfactory, and the most recent surgical writers even are hopelessly confused in their descriptions of these obscurer forms of injury. There has been no lack of tabulated collections of cases, but they have been disjointed and heterogeneous, incomplete in historical detail, and barren of result for any purpose of useful generalization. They have presented a jumble of symptoms, lesions, and pathic relations, at once perplexing and discouraging. I limit criticism to methods of culture which have obtained in the field of traumatism, and have no intent to disparage the work which has been done in other departments of neuro-pathology.

I have no hesitation in ascribing this want of precision primarily to an erroneous conception of the structural alterations which such traumatisms produce, and to a consequent failure to either accurately define the resultant morbid conditions or to systematize the symptoms which they present. Following an imperfect apprehension of the nature and effects of structural lesions, intracranial injuries have been considered largely in the light of theoretical preconceptions. Mistaken views of both pathology and symptomatology, strengthened by time and tradition, have retained acceptance, or have been formally discarded, only to be again practically rehabilitated. Recognition is still given to a hypothetical disorder which is without pathological foundation; symptoms are still grouped under a single comprehensive designation, which result from varied pathogenic conditions, and which present as many points of contrast as of similitude; fact has been subordinated to fancy in order to establish antitheses which are inaccurate in every particular; a comprehensive inflammation of the entire cranial contents has been assumed which has no basis of truth. Concussion, compression, and encephalitis are terms which still hold a place in the vocabulary of surgical literature. I have heretofore considered the subject of concussion, and shall recur to compression and encephalitis hereafter.

In a previous paper, of which this is a continuation, and to which I am so often compelled to refer, I detailed the traumatic lesions which were revealed in a considerable number of necropsic examinations, and made them the basis of classification of the morbid conditions which they had occasioned. In an even larger number of necropsies observed since that time parallel conditions have been found to exist. As each form of lesion is attended by characteristic symptoms, and as no evidence is adduced that symptoms occur independent of anatomical alteration, it is logical and, I think, essential to recognize groups of symptoms under the name of their pathogenic lesion. The attempt to classify traumatic or other diseases by their outward manifestations is arbitrary, misleading, unphilosophical, and contrary to what has come to be accepted as the true principle of nosography. It is beyond my province to insist upon the proper basis of nosology, which has been so learnedly demonstrated and so felicitously formulated by my distinguished colleague, Dr. J. W. S. Gouley.*

---

* *Diseases of Man—Nomenclature, Classification, and Genesis.* Dr. John W. S. Gouley, Surgeon to Bellevue Hospital, New York, 1888

These lesions, reaffirmed in brief, are: (1) Intracranial hæmorrhages from injury of the bone, brain, or membranes; (2) arachnitis, from injury of the arachnoid membrane and pia mater; (3) lacerations and contusions of the brain substance; to which may be added (4) pyogenic parenchymatous inflammation.

I omit reference to disorganizing injuries in which the brain and its membranes are alike involved; they are patent and have no relation to classification. There is another lesion—thrombosis of the dural sinuses, which I have been unable to connect with symptoms.

The prevalence of errors in pathology and of faulty generalizations in symptomatology, together with the inherent force which they derive from prescription, may be reckoned extrinsic causes of diagnostic uncertainty. Some of the sources of confusion and failure which I indicated in a previous study of head injuries may be regarded as intrinsic. In the second class the multiplication of lesions in the same case and the apparent identity of symptoms from dissimilar causes may be counted as most efficient. I believe the "Ariadnean thread" should be sought in the study of those cases in which the lesion is either simple or in which one out of many is primary and of paramount importance. The clew once gained, it ought to be possible to follow it through the more complicated cases.

## I. HÆMORRHAGE.

Traumatic intracranial hæmorrhages are usually classified as (1) epidural, (2) subdural, and (3) cortical or pial. This is hardly accurate or complete, though not absolutely objectionable. No exception can be taken to the term epidural as denominative of an extravasation between the bone and the dura mater, for it is anatomically correct. The use of the word subdural is less felicitous, since the effusion is into the arachnoid cavity and not between the dura and the parietal arachnoid. Hæmorrhages, again, which occur between the visceral arachnoid and the brain are better subdivided so as to imply cause as well as location. A more accurate classification of hæmorrhages, if made purely in accordance with location, would be: (1) Epidural, (2) arachnoid, (3) subarachnoid; but as the arachnoid variety is merely an accidental extension of any subarachnoid hæmorrhage, and as the subarachnoid is of composite origin, a better, and I believe the best possible, subdivision is into (1) epidural, (2) pial, (3) cortical.

The matter of nomenclature is of absolute importance, since pathological exactness is essential to correct diagnosis, and a change of established form, always to be deprecated, becomes in this instance a logical necessity.

I at one time believed epidural hæmorrhage to invariably result from cranial fracture. I have more recently seen three cases in which it was occasioned by contusion from *contrecoup.* Its source may be either in the diploe or in the osseo-dural vascular connection. Pial hæmorrhage is caused by contusion of the pia mater and consequent rupture of its vessels, while cortical hæmorrhage, though occupying the same anatomical position in the subarachnoid spaces, is derived from laceration of the brain surface. These three hæmorrhages are always primary; arachnoid hæmorrhage, as I have stated, is always secondary. It is in most cases a cortical hæmorrhage which breaks through the pia and visceral arachnoid; a pial hæmorrhage from contusion is not often in sufficient amount to rupture the arachnoid membrane, though an occasional instance will be found in the histories which I have presented. That blood from the osseo dural vessels may reach the arachnoid cavity when the injury at the same time involves both bone and dura is evident, but that no epidural hæmorrhage in itself has power to rend the dura seems equally certain; the fibrous structure resists while the brain substance is compressed and displaced, even though blood is effused in enormous and fatal quantity.

Intracerebral hæmorrhage is the result of subcortical laceration, or rather a part of it, and therefore not a distinctive lesion.

The differential diagnosis of hæmorrhage is of special importance, since of all the intracranial lesions it most frequently admits of operative interference. Its origin and location are no less important as constituting a second factor in determining the propriety of operation.

A casual examination of the cases which I have recorded will demonstrate the exceeding frequency of hæmorrhage in all forms of intracranial injury. In nearly sixty per cent. it has occurred in sufficient quantity and in such relation as to largely influence the final result, and to become a more or less determinate factor in the genesis of symptoms. In one third of this percentage it has been the direct and probably the sole cause of a fatal termination. It is doubtful, however, if it is ever an isolated lesion. In a very large proportion of the whole number it was secondary to laceration, and while this was in itself often insignificant, the hæmorrhage was none the less profuse and the source of both symptoms and danger. In the residue of cases, though it was primary, it was not unexpectedly associated with other structural alterations. The same violence which is sufficient to separate the dura from the bone, or to rupture the vessels of the pia mater, can hardly fail to be transmitted to the brain, and its effect either concentrated in a local laceration by *contrecoup* or diffused in a general contusion of its substance. A hæmorrhage is often regarded as uncomplicated from want of sufficiently careful necropsic inspection of the brain throughout its whole extent. There may be no laceration or other obvious local injury, and general contusion is readily overlooked.

I have but once observed in necropsy a hæmorrhage where the associated cerebral contusion seemed so slight as to be unimportant. There are twenty or more cases, however, in which hæmorrhage was the essential lesion, and which, perhaps, afford sufficient ground for inductive examination. There is probably a larger number than I shall analyze, but it is impossible to rate them with even approximate precision. They include nine epidural hæmorrhages and eleven of pial or cortical origin, of which five had reached the arachnoid cavity. If to these are added eight cases which were subjected to trephination, and in which the existence and location of hæmorrhage was thus verified by operation, and in which no considerable depression of bone or other evident complicating lesion existed, the total number will be increased to twenty-eight. One of the operative cases disclosed both epidural and arachnoid hæmorrhage, and terminated in death; the others were all of epidural character, and resulted in recovery.

The symptoms ob-erved were not numerous, and of these temperature, when considered with proper regard to its surrounding conditions, was of greatest diagnostic significance. In seven of the necropsic cases it was unrecorded; in seven of those remaining it was on admission subnormal; in five it was 99° to 99°+; and in one, which also involved slight contusion of the corpus striatum, it was 101°. In those cases, three in number, in which it subsequently exceeded 101°, there were notable coexistent lesions of the brain substance; one presented extensive lacerations of the base, another extensive general hyperæmia and œdema, and in a third, in which the temperature rose from 94° to 102° in the eight to ten hours which preceded death, there was implication of a supposed heat center. In the operative cases the highest temperature was 101·6°, and in the only one in which it exceeded that degree it reached 102° after the formation of a fungus cerebri. I have already attributed the early subnormal temperature to shock, and, when the patient has survived this condition, I have seen that the temperature has been restored with the general reaction. In each case in which subsequent elevation exceeded 101°+ there has been marked general contusion or other concomitant injury of the parenchyma of the brain. In these twenty eight cases, therefore, best fitted for observation, the temperature characteristic of hæmorrhage has been found to range from above normal to 101°+.

The one constant symptom in fatal cases was some degree of unconsciousness. In the majority it was profound, or at least complete, from the moment of injury to the end of life. In four others consciousness was primarily lost, and, after more or less complete restoration, was merged in final coma. In three instances consciousness was retained for some length of time, during which the patient walked for a considerable distance, and then either gradually or suddenly became unconscious. In another case of late unconsciousness delirium followed, and continued till death occurred. In a final instance unconsciousness was primary, but, as in the case just mentioned, delirium followed hard upon it without a period of conscious intelligence. In the operative cases in which recovery ensued, and in which it is fair to assume that the effusion was smaller, loss of consciousness was less constant, occurring in but half their number. In two the mental condition remained unaffected, and in one unconsciousness was replaced by delirium; in three cases in which it was a symptom, it was very transitory in two, and in one but moderately prolonged.

The varying phases of unconsciousness, the diverse symptomatic conditions with which it is associated, and the uncertain period of its occurrence, render it impossible to accept the traditional explanation of its existence, that it is solely dependent upon a mechanical compression of the subjacent brain substance. It is probable that as a primary symptom—as an instantaneous result of injury—it is due to general contusion, which is itself an instantaneous lesion. It has been seen in the larger number of the fatal cases collated that it has been absolutely the first symptom, not only at the time of admission, but as learned at the scene of accident and noted in the ambulance history. The effusion of a sufficient amount of blood to act mechanically requires an appreciable interval. This is evident in two of the cases of rupture of the arteria meningea media, in which some hours elapsed before the patient became unconscious, and in which the epidural clot was found to be of enormous size. There may or may not be a restoration of the intellectual faculties between the earlier and the later lapses of consciousness. The general cerebral contusion may be so severe that the unconsciousness which it produces will continue till the effusion has become sufficient to occasion the same condition as a direct result, and one is lost in the other. It is also possible that the central lesion may be insufficient to annul consciousness for the time necessary to the effusion of blood in sufficient quantity to act as an immediate stupefying agent. This opinion as to the manner in which loss of consciousness occurs in intracranial lesions will be strengthened by the wider comparison of cases to be made, in which hæmorrhage was a contributive rather than an essential lesion, and in the direct study of other forms of injury.

Much importance has been attached to disturbance of the pupils in traumatic hæmorrhage. The cases under present consideration in most instances show some change in the pupillary condition. It has been unnoted in two of those which were fatal and in three of those which were subjected to operation; it has been normal in but three out of the remaining twenty-three. The pupils in the cases of abnormity have afforded almost every possible combination of dilatation with contraction. In six both pupils were dilated, the hæmorrhage being in three epidural, in two pio-arachnoid, and in one epidural and pio-arachnoid combined. In four both pupils were contracted, the hæmorrhage being in one epidural, in one pial, in one both epidural and arachnoid, and in one both epidural and cortical. In two the pupil was dilated on the side of injury and contracted on the opposite side, the hæmorrhage in each being epidural; in two the pupil was contracted on the side of injury and dilated on the opposite side, the hæmorrhage in each being epidural and derived from the middle meningeal artery; in three the pupil was dilated on the side of injury and normal on the opposite side, the hæmorrhage in each being epidural; in three the pupil was normal on the side of injury and dilated on the opposite side, the hæmorrhage being cortical in two and epidural in one. There was no instance of contracted pupil on either side without change in its fellow. In the three cases in which both pupils remained normal the hæmorrhage was epidural in one, pial in another, and cortical in the third. The hæmorrhages occurred upon every part of the cerebral and cerebellar surfaces, vertex, and base.

There seems to be no change in the pupils, Hutchinsonian or otherwise, which is positively characteristic. In two thirds of the cases analyzed the hæmorrhage was wholly or in part epidural, and in two thirds of these again one pupil or both was dilated; but as in the aggregat sorts of pupillary changes resulted from all sorts of hæ rhages, their observation can be scarcely more than confirmatory of an opinion justified by the collation of other symptoms. Their condition as to mobility was scarcely more to the purpose; in the far greater number, whatever the origin, location, or amount of hæmorrhage might be, they were freely movable.

The pulse was unnoted in three cases; it was normal in four, in two of which the hæmorrhage was epidural, covering the convex surface of a hemisphere, and in two was of subarachnoid origin, occupying the inferior occipital fossæ. In the larger number of cases it was frequent, and the hæmorrhage, usually large, was of either variety and variously situated. In six cases in whch the pulse was slow, the hæmorrhage was in each instance epidural, and the patient profoundly unconscious, and in four the respiration was stertorous. In neither the fatal nor the operative cases was there any definite relation discovered between the character of the pulse and the nature of the hæmorrhage, or between it and the associated symptoms.

There is another pulse condition, a want of symmetry in radial pulsation upon the two sides of the body, which I have found to occur in connection with both hæmorrhages and visceral injuries, and shall give consideration hereafter.

The respiration afforded more definite indications. It was normal in but a single instance. It was increased in frequency in two cases moderately, and very markedly or excessively in six others, the hæmorrhage having occurred with a single exception upon the convex surface of the brain. In nine cases the respiration was stertorous, and in seven of them the hæmorrhage, which was epidural in six, not only covered, but compressed the cerebral surface upon the side of injury or occupied the anterior fossæ; in the other two it was rapid, accompanied by cyanosis and pulmonary œdema, and the hæmorrhage covered the pons and to some extent the medulla. None of the operative recovering cases presented any noticeable deviations from normal respiration.

The disturbance or abrogation of muscular function was an occasional symptom, and was exhibited in accordance with established laws of cerebral localization. Paralysis occurred in three of the fatal cases and in two of those which recovered after operation; it was hemiplegic in four and paraplegic in one. Muscular rigidity, affecting one side or both, occurred in five cases, and general convulsions in one which was fatal. In each case some part of a motor area was covered by the hæmorrhage, which was indifferently epidural, cortical, or pial, and acted as a paralyzing or irritant lesion according to its extent and situation. These motor disturbances, while of great positive diagnostic importance, are so frequently absent that they have no corresponding negative value.

In a single case there was protrusion of both eyes as well as dilatation of both pupils. There was found an epidural clot in the right inferior occipital fossa and an arachnoid hæmorrhage which covered both frontal, and the parietal lobes as far as the fissure of Rolando.

Sensory disturbances were still more infrequent. Delirium was noted in three cases: in one which recovered it was primary, and the hæmorrhage, as disclosed by the trephine, was epidural and in trivial amount; in two fatal cases it was of later occurrence, followed a previous condition of unconsciousness, and was associated, in one with a pial hæmorrhage over the right hemisphere and inferior surface of both occipital lobes, and in the other with a cortical hæmorrhage covering the pons; in both general contusion was well marked. Partial anæsthesia, irritability, and restlessness were observed in isolated cases.

In order to further test the diagnostic value of the symptoms observed in this limited number of cases, I have analyzed thirty-four others in which, though the associated lesions were more severe, the hæmorrhage was sufficiently large, absolutely or relatively, to be a probable source of distinguishable symptoms. They present some points of difference which naturally followed from different attendant conditions. In the larger proportion of both necropsic and operative cases, in which hæmorrhage seemed to be the single source of danger, it was of epidural origin. In the present group of cases, in which the brain and its membranes are more seriously involved, it is with few exceptions essentially pial or cortical. When these parts are the seat of excessive general contusion without laceration, the pial vessels are naturally the ones most likely to suffer rupture, and in fact in every such instance the hæmorrhage, if subdural, was of this character; in two it chanced to be epidural. When the brain substance is superficially wounded, the cortical vessels are obviously most likely to be the source of hæmorrhage. It is also inevitable that when life is prolonged the symptoms of hæmorrhage should be often modified, superseded, or complicated, by others characteristic of the additional lesions.

The temperature loses its diagnostic importance. It is generally higher than in the previous instances where hæmorrhage was less complicated. In ten cases it ranged from 105° to 107·8°, and in twenty-six it was above 103°. In the cases which terminated fatally within twenty-four hours, which was the limit of life ascribed solely to hæmorrhage, the temperature, as in them, did not usually exceed 101° + ; in four, however, in which death occurred within even less than twelve hours, it rose to 102·2°, 106·8°, 107·8°, and 103°. Subnormal temperatures on admission were infrequent.

Consciousness in these cases, as was noted in those subjected to operation, was less uniformly lost than when death seemed to result directly from hæmorrhage, yet in far the larger number its loss was primary, complete, and permanent. In some it was at first partial, but progressive, and eventually complete; in others primary unconsciousness merged in delirium; in a few instances consciousness was at first retained, only to be lost at a later period. In general, the results of this examination are confirmatory of those obtained from the study of the less complicated cases.

The pupillary condition was less diversified than in the cases previously detailed. It was normal in about the same proportion of those in which record was made. There was much more frequent dilatation of both pupils—more than twofold; an equal number in which both were contracted, and consequently fewer instances in which the two presented opposite conditions. As before, there was no case in which one pupil was contracted without change in its fellow. When both pupils were abnormal the hæmorrhage was usually bilateral; and in unilateral dilatation the hæmorrhage was usually upon the corresponding side; but in neither instance was the rule invariable. In the two cases of normal pupils the hæmorrhage, which was large in each, was epidural in one and pial in the other, and in each was associated with important change in the brain substance.

The pulse when registered was, perhaps, under the influence of opposing forces, usually normal. It was occasionally slow or unduly frequent, but oftener exhibited that want of symmetry in force and fullness upon the two sides which I have mentioned as occurring in different forms of intracranial injury.

The respiration was unnoted in a third of the cases, and in many of these, which were among my earlier observations, it was doubtless unaffected, since at that time normal conditions were unrecorded. If moderate allowance be made for such omissions, the proportion of mixed cases in which its frequency was from 18 to 24 in the minute, and in which it was without special characteristics, was from one third to one half, while in those in which hæmorrhage was more nearly an isolated lesion it was of normal character in but a single instance. It was stertorous in about the same proportion of cases as in the former class, so that those remaining, in which it was abnormally slow or frequent, are necessarily few.

The muscular system again not infrequently afforded symptomatic indications. In each instance in which an irregular excitation of functional activity was manifested by either clonic or tetanic contraction the hæmorrhage was complicated by cerebral or cerebellar laceration. In others, in which muscular power was lost or held in abeyance, the complicating lesion was invariably general contusion. Clonic contractions were relatively frequent; general convulsions, while but once observed in the class of comparatively pure hæmorrhages, and then as merely localized convulsive movements, occurred in six of the mixed cases. General muscular rigidity in the two classes occurred with more nearly equal frequency. These facts are suggestive of the influences exerted by different lesions.

Since, in the group of cases under consideration, the hæmorrhage is in each instance associated with some serious injury of the immediate seat of sensory and intellectual function, symptoms which depend upon disturbance rather than upon simple oppression of the nerve centers are to be regarded here as only indirect. Delirium, irritability, or restlessness, when of immediate occurrence, and the effusion of blood is moderate in amount, may be considered symptoms of hæmorrhage, but only in the sense that a pleuritic pain is counted a symptom of pneumonia. It is unnecessary, therefore, where direct brain injury is a recognized factor, to investigate such conditions while engaged in the study of uncomplicated hæmorrhages.

There are two symptoms which have been often held to be diagnostic of intracranial hæmorrhage: these are loss of consciousness following cranial injury after some appreciable interval, and dilatation of the pupil. This view is not well sustained by the statistical facts which I have collated. Reference to either group of cases will disclose comparatively few instances in which consciousness was lost in the manner indicated. Some change in the pupillary condition was found to occur in most of them, but it was varied in character and not to be regarded as typical in any one of its forms. Various other symptomatic manifestations have been suggested as indicative of this particular lesion. Some of them, like the dilated and insensible pupil, occur often enough to afford corroboration of an opinion founded upon other evidence; others which are possible, but, in fact, infrequent, are given an exaggerated diagnostic importance; and others still, when they chance to exist, have no relation to hæmorrhage. The absolute value to be attached to these reputed pathognomonic symptoms can be only determined by a reference to the results of actual observations in such an extended series of cases as I have in this instance collected.

### II. SUBARACHNOID SEROUS TRANSUDATION.

In place of a hæmorrhage a subarachnoid serous effusion is sometimes encountered which is not of inflammatory origin. I first called attention two years ago to a special contusion of the membranes as the immediate cause of traumatic arachnitis. Such a contusion may occasion either a simple meningeal hyperæmia, a pial hæmorhage, a dropsical subarachnoid serous effusion, or some grade of meningeal inflammation.

The dropsical transudation is not of frequent occurrence, and perhaps not easily distinguished from a low grade of inflammatory exudation. If it occurs over limited areas without subarachnoid clot, and without opacity of the arachnoid membrane, or other evidence of the inflammatory process, there would seem to be little doubt of its nature. All these conditions rarely concur, but there are at least three such instances in my later series of cases, and in two of them the very early fatal termination, within a few hours only, corroborated the opinion formed from necropsic examination. This œdema, like simple hyperæmia, is found to exist in the presence of more serious organic changes, so that it is impossible to connect it with symptomatic conditions.

### III. ARACHNITIS.

The impression conveyed by many surgical writers is that traumatic arachnitis is of rather frequent occurrence, and a constant menace in the convalescence of all cases of intracranial injury. My personal observation has led to a different conclusion. If serous effusions of positive or probable congestive origin, and a limited number of meningeal inflammations which were without apparent influence in the progress of other and directly fatal lesions, are excluded, there remain but thirteen cases in which arachnitis was undoubtedly existent and at the same time influential in compassing the final result. These comprised six in which the effusion was purulent, and seven in which it was serofibrinous, in one of which the presence of the *Streptococcus pyogenes* was demonstrated by culture. In one of the acute cases the purulent formation was the extension of a more profuse pyogenic process along the course of a drainage-tube which traversed the brain substance, and in another it was an equally direct extension from an infected compound fracture which lacerated the membranes and cerebral cortex. In both, the symptoms were merged in those of the primary lesion. The number of cases useful for purposes of analysis is thus reduced to eleven, in none of which the inflammatory process seemed to be propagated from a localized injury of the brain or membranes. They are too few in number to afford a basis for any wide generalization in either symptomatology or diagnosis. The course of traumatic arachnitis, however, is not unlike that of the idiopathic form, and conclusions are thus less dependent upon direct observation than in the study of other intracranial lesions. These cases are at the same time in sufficient number to make their analysis important and to justify an epitome of their already abstracted histories.

Case I. *Acute.*—Primary symptoms referable to depressed fracture, subcortical laceration, and general contusion. On the second day, patient irritable and somnolent. Temperature, 102°; bone elevated. Fourth day, temperature, 105°. Fifth day, patient delirious, restless, and sensitive to external impressions; surface hot, pupils moderately dilated and slow to act, and coma which continued till death on the seventh day. Temperature varied from 104°+ in the morning to 105°+ in the evening; pulse and respiration much increased in frequency.

*Lesions.*—Fracture of base and vertex; lacerations, pial hæmorrhage, and contusions of brain and membranes; purulent effusion over occipital and posterior parietal lobes on both sides, and in inferior occipital fossa of the side of injury.

Case II. *Acute.*—No primary symptoms. On second day, temperature, 101·6°; and afterward 99°+ till the tenth day, when without the occurrence of intervening symptoms it rose to 101°+, on the next day to 104·6°, and afterward varied from 103° to 104°+ till the fifteenth day, when it began to rise progressively and reached 107·4° on the sixteenth day shortly before death. On the tenth day, coincident with the rise in the temperature, the patient became restless and slightly delirious; on the eleventh day he had a slight chill and increased delirium; on the fourteenth day there was posterior cervical rigidity, and on the fifteenth a slight general convulsion. The pupils and respiration were normal, pulse was 104 to 112, and mental condition sluggish.

*Lesions.*—Fracture of vertex which was confined to external table; purulent effusion over both frontal lobes, encroaching upon parietal and extending into median fissure.

Case III. *Acute.*—Patient in alcoholic condition; restlessness succeeded by stupor, occipital pain, with epigastric pain and vomiting. Temperature on admission, 102·6°; next day, 105°; and third day, 103·8°; pulse moderately accelerated, 120 to 84; respiration not above 24, and pupils normal.

*Lesions.*—Subarachnoid serous effusion; much purulent effusion anteriorly upon both sides of the vertex, but mainly upon the left.

In each of the previous instances the presence of the streptococcus was demonstrated by culture.

Case IV. *Subacute.*—Primary symptoms from subcortical laceration and general contusion, which subsided through second and third weeks. Temperature fell to below 100°, and patient became asthenic. Three days before death he became unconscious, and the temperature rose to 100°+, and on the next day to 103·8°; then varied from 103° to 104·8°, and later rose progressively to 109°. There were alternating periods of rational intelligence and of wild delirium up to the time of final unconsciousness.

*Lesions.*—Subcortical laceration and excavation of an entire frontal lobe, and excessive general hyperæmia; serous effusion over all parts of the brain with well-marked arachnoid opacity.

Case V. *Subacute.*—Primary symptoms of general contusion and of lesion of the motor area. Fourth and last week, mental apathy replaced by mild delirium; three days before death temperature rose from 99° to 104°, declined to 100° on the following day, and again rose to 104°. Death occurred on the twenty-eighth day.

*Lesions.*—General contusion and a small superficial cavity from a local contusion in the leg area of one side; serous effusion compressing both frontal lobes.

CASE VI. *Subacute.*—Primary symptoms of cerebral injury; temperature did not exceed 99°+ for the first week, then rose suddenly to 102·8° to 103·2°, and afterward varied irregularly from 99° to 104°; pulse and respiration only became frequent at the last; mental condition alternately stupid and delirious from the beginning. Death occurred in sixteen days.

*Lesions.*—Fracture of the base, laceration of both frontal, and of one temporo-sphenoidal lobe; subarachnoid clot in occipital region; general serous effusion with arachnoid opacity.

CASE VII. *Subacute.*—Primary symptoms from cerebral contusion which continued three weeks. Temperature in second and third weeks, 98·5° to 99°+, with progressive mental improvement. In the fourth week the mental condition deteriorated. On the twenty-third day, temperature, 101°; in the four days following it varied from 98·5° to 101 6°, and the pulse was frequent; on the twenty-seventh day, temperature rose from 100·6° to 106·6° without remission, and in the right axilla was from half a degree to a degree and two tenths higher than in the left; on the twenty-eighth and last day it was 108°, and the pulse, previously frequent, was from 54 to 74. The respiration was accelerated during the last two days, and the patient became irritable and restless.

*Lesions.*—Fracture of the base, several necrotic contusions upon the superior surface of the prefrontal lobes; general contusion with œdema; and general serous effusion over superior and lateral surfaces of the brain.

CASE VIII. *Subacute.*—Primary symptoms from parenchymatous injury. Temperature normal from sixth to ninth day, and afterward much of the time subnormal till the thirteenth day, when a severe chill was accompanied by an elevation to 101°+. A second chill on the nineteenth day was followed by a progressive rise of temperature to 105·5° at death on the twenty-first day. After the second chill the patient grew mentally sluggish and became weaker; pulse and respiration were frequent.

*Lesions.*—General contusion of the brain with œdema; sub-cortical laceration of a frontal lobe; and serous effusion with arachnoid opacity over superior and lateral cerebral surfaces

CASE IX. *Subacute.*—Primary symptoms, those of general contusion, followed by an intercurrent bronchitis. On the thirteenth day, occipital headache, which became general; somnolence and irritability; temperature, 101° to 104°, and on the eighteenth day, 105°, with delirium and post-cervical rigidity; later, increased delirium and somnolence, with symptoms referable to implication of cranial and spinal nerves; progressive emaciation; lack of urinary and fæcal control; rapid and insufficient respiration; unconsciousness and death on the thirty-first day. Temperature from 100° to 104°; pulse, 64 to 90; axillary temperatures variable, and when unsymmetrical, more frequently half a degree higher on the side opposite original injury.

*Lesions.*—Fracture of the base, thrombi in the lateral sinuses, general contusion of the brain with œdema; turbid serous effusion in the ventricles and at the base; thick membranous effusion over the pons, medulla, inferior surface of the cerebellum, and in the fissure of Sylvius; fornix much softened, and trivial lacerations of one temporo-sphenoidal lobe. The *Streptococcus pyogenes* was discovered in cultures of the membranous effusion.

CASE X. *Subacute.*—Primary symptoms of general contusion; unconsciousness which merged in final coma; restlessness; pupils widely dilated but responsive to light; urine retained; left side temporarily rigid; right side paretic; pulse and respiration continuously frequent; temperature on admission normal; subsequent variations: 102·2°, 101°, 101°+, 102°, 105°, 104°+, 102°+, 100°+, 101°+, 102°+, 105°+, 106°: one hour post mortem, 106·2°. The axillary temperatures were symmetrical for forty-eight hours; then, with a rectal temperature of 102·6°, temperature in the right axilla was 103·2°, while in the left it was 100·4°; and at fifteen minutes' intervals rose to 101·4° and 102·8° in the left without change in the right. At this time the left upper extremity and foot were intensely cold to the touch. Death occurred in fifty-three hours.

*Lesions.*—General contusion with œdema; laceration in substance of the fornix; small pial hæmorrhage upon the inferior surface of the cerebellum; small hæmorrhagic serous effusion in one lateral ventricle, and large serous effusion with arachnoid opacity over the occipital and posterior portion of the parietal lobes.

CASE XI. *Acute.*—Primary symptoms of fractured base; temperature high from the first day; from 104°+ to 105·6° till fourth and fifth days, when it receded to 102·8° to 101°, and again rose to 104°+ to 105°+ on the sixth and seventh days; muscular twitchings of the right side of the body and left hemiplegia and hemianæsthesia on the sixth day; general convulsion beginning on the right side preceded death, which occurred on the seventh day.

*Lesions.*—General subarachnoid purulent effusion most copious over the left frontal lobe below the origin of the fracture.

It would seem impossible to determine *a priori* the circumstances under which an arachnitis is likely to follow meningeal injury. The alcoholic habit existed in but a minority of cases, the previous constitutional condition was often unimpaired, and the age ranged from early youth to past the middle period of life. The coexistent lesions were diverse, and had no obvious relation to the changes which the membranes had suffered. Fractures of the skull, cortical or subcortical lacerations, hæmorrhages, or notable general contusions were variously discovered upon necropsic examination. Some degree of meningeal implication is probably almost invariable in intracranial injuries, but these cases fail to afford a clew to the immediate conditions which occasionally favor the development of arachnoid inflammation. The time of invasion was equally uncertain; it was in some instances immediate, and in others delayed for weeks after the reception of the injury. The interpretation of symptoms had therefore to be made without material aid from considerations of time or circumstance.

In three cases the arachnitis was primary, in another its initial symptoms were so insidious as to fail of recognition, and in the remaining seven its invasion was late and sharply defined. In the larger number, which may be considered typical, the course of symptoms referable to complicating lesions was interrupted by a distinct and somewhat sudden elevation of temperature accompanied by an evident change in the general condition of the patient. He became irritable, restless, delirious, or somnolent, and in one instance suffered a severe chill, though the effusion did not prove to be of purulent character. The subsequent range of temperature was erratic. It was marked by variations from day to day or from hour to hour, not usual in other intracranial lesions. The arachnitis was so constantly associated with other grave structural alterations that it is impossible to demonstrate its exact relation to temperature, but if my observations in these few cases which were capable of verification may be supplemented with others made in recovering cases which I had reason to believe were arachnoid inflammations, I should infer that this variation was characteristic and ranged from 101°+ to 104°+. In the majority of verified fatal cases the temperature immediately before death was from 105°+ to 109°; but in each some lesion of the brain substance existed in which a very high temperature was to be expected. In one exceptional case without such complication, in which it reached 107·4°, the effusion was purulent. Whether or not these fluctuations of temperature are due to a secondary implication of thermotaxic centers situated in the cerebral cortex, as suggested by Hale White, is immaterial in a study of symptoms as related to diagnosis.

After the invasion, and aside from peculiarities of temperature, the progress of the diseases was especially characterized by continued manifestations of cortical irritation. Some grade of delirium persisted in almost every case, and restlessness, irritability, or extreme sensitiveness to external impressions was often marked long after consciousness was finally lost. General or post-cervical muscular rigidity, in one instance a slight general convulsion, and in another a chill, were further indications of nervous excitation. They all, with the exception of the case of basilar inflammation, terminated within the week, and rather from asthenia than from coma the result of pressure. The pupils were oftener normal than otherwise, and the pulse and respiration failed to reflect the existing inflammatory process; moderate acceleration of the pulse and very slight, if any, increase in the frequency of respiration seemed to be the usual conditions. It can not be said that there was any sharp contrast in symptoms which indicated the character of the effusion. In one of the acute cases there were classical symptoms of sthenic inflammation, but there was no chill; in another, which began with a chill, the subsequent symptoms were no more pronounced than is common in the subacute form; while in the third and last, the invasion and progress of the inflammation were remarkably insidious.

The question of infection is uncertain. There was fracture of the vertex in two of the acute cases, and a scalp wound in the third; and in the case of basilar arachnitis, in which the *Streptococcus pyogenes* was discovered in a large sero-fibrinous effusion, there was a fracture through the internal auditory canal and rupture of the tympanum. There was no more than a possibility of direct infection in three out of the four; the wounds were maintained in an aseptic condition, and in the absence of an evident pyogenic process there was no proved pathogenic relation between the external lesion and the character of the internal inflammation, and no good reason to assume that it existed; if in the fourth case the access of the pyogenic germ is more readily comprehensible, the general history is better interpreted upon the supposition that its development was in the usual course of idiopathic secondary serous inflammations in prolonged disease, with the added predisposition derived from a previous local contusion.

I am indebted to the courtesy of Dr. H. M. Biggs for notes of eight unpublished cases of infective purulent arachnitis which were not of traumatic origin. They exhibit the same irregular fluctuations of temperature and the same varied manifestations of cortical irritation which were observed in traumatic cases of either form which I have collated. They are of interest here as confirmatory of the proposition that the symptoms of arachnitis are not necessarily modified by its cause or grade.

It is clear from this analysis of meningeal hæmorrhages and inflammations that the attempt to crystallize their symptoms with those of depressed fractures, and to formulate in a single word—compression—a resulting condition, is futile and misleading. It is no more defensible than a former practice of grouping organic diseases under the common name, dropsy. These several results of cranial injury indicate entirely different pathic conditions, and their external manifestations are more marked in their differences than in their resemblances. Even the cerebral compression which they are supposed to characteristically produce is in the majority of cases absent or replaced by a still more characteristic irritation. When it is further attempted to discriminate these artificially consolidated lesions from injuries of the brain substance by antithetical tabulations of symptoms, the possibilities of error are arithmetically increased. It is practicable from an examination of the cases which I have cited to demonstrate the unreliability, both positively and negatively, of each assumed individual diagnostic symptom in any one of the tables which are scattered through surgical text-books. The importance of a statement which I have previously made that a proper classification of morbid conditions must be based upon structural alterations, and their diagnosis established by careful analysis and comparison of resulting symptoms, is warrant for its repetition. The diagnosis of fractures, meningeal hæmorrhages, and meningeal inflammations from brain lesions in the manner to which I have excepted fails in both particulars. A defective classification has been supplemented by an inaccurate analysis of symptoms, and faulty generalization has resulted in prevalent confusion.

### IV. LESIONS OF THE BRAIN SUBSTANCE.

The injuries which the brain may suffer are general and local: a diffused contusion or a limited lesion, which may be either a contusion or a laceration. The analysis of cases which I have made demonstrates the exceeding frequency of visceral lesions. I have intimated the probability that some degree of general contusion always exists in intracranial injury, even though some other lesion may be paramount. At the time of my earlier observations it was only noted when its evidences were strikingly apparent, and even at a somewhat later date only when they were more than ordinarily well pronounced; yet with these limitations it will be found recorded in more than fifty per cent. of the total number of cases. Local contusions, on the contrary, have been of comparatively infrequent occurrence, while lacerations have been discovered even oftener than well-marked structural alterations of a general character. Meningeal contusion, independent of recognized visceral injury, has been encountered in but three instances, in each of which a fatal arachnitis resulted. Epidural hæmorrhage, in which the effects of violence inflicted upon the cranium had not been extended to the brain, has not been once disclosed. Some implication of the brain, therefore, may be regarded as practically assured in all cases of cranial injury.

In the study of brain lesions it is necessary to determine not only the symptoms they may have in common, but if possible the existence of others characteristic of individual forms.

1. GENERAL CONTUSION.—Notwithstanding the very constant occurrence of general contusion, it so rarely terminates fatally when uncomplicated by other structural changes that opportunity for observation of its distinctive symptoms is much more limited than in cases of hæmorrhages and arachnites. I am enabled, however, to present six cases in which no concomitant lesion existed, or in which, if present, it was so trivial that it may be fairly assumed to have had no influence in the production of symptoms. In one there was absolutely nothing beyond the general contusion; in two there were also limited and non-infective dural thromboses; in another there was a single small extravasation into an optic thalamus, and in the other two there was a slight cortical laceration and correspondingly unimportant cortical hæmorrhage. In all there was a more or less intense general hyperæmia, which was sometimes more strongly pronounced in some particular region, as anteriorly, posteriorly, at the base, or in one hemisphere, than elsewhere. In three cases the pia was notably engaged; in three there was well marked or even excessive general œdema; in four, thrombosis of the minute vessels, which generally characterizes contusion, was a pronounced feature. I regard the last-mentioned condition as a manifestation of contusion, as it is habitually absent in the hyperæmia of idiopathic disease. Punctate extravasations were less numerous than is usual in the more frequent instances in which hyperæmia is associated with laceration. In those cases in which

death was long deferred, the absence of inflammatory processes was verified by microscopic examination made at the time of necropsy.

The analysis of symptoms in the six cases is unsatisfactory. The few connecting links which measurably held together the cases of hæmorrhage or arachnitis have no corresponding representation. There was no uniformity either in the occurrence of individual symptoms or in their course or termination. In the single one which was absolutely uncomplicated there was no loss of consciousness at any time, till its final lapse from asthenia; in all the others it was primary and in three was permanent. There is no other individual symptom which occurred in more than half the cases cited. The pupils were dilated, contracted, or normal; the pulse and respiration were variable. It is true that delirium, mental irritability, or apathy, combined with muscular rigidity, convulsions, or some degree of paralysis, occurred in each instance save one, and in that one a profound coma from the beginning held in abeyance all mental and motor functions; but the time of their appearance and the method of their combination had no conformity to rule. Headache, persistent vomiting, and perforating ulcer of the cornea were isolated phenomena, and in one protracted case dementia preceded death.

The temperature again probably affords the earliest indication of the intracranial condition. It was never subnormal on admission, and was never more than moderately elevated; in four cases out of five it was from 99° to 100°; in the fifth it was 101°+, as it was in the sixth, in which it was not recorded till the second day. Its subsequent course was in general progressive, and with one exception attained a high degree before death ensued. Recessions were observed only once or twice in two cases which were considerably prolonged.

It is not difficult to comprehend the reasons for the diversity of symptoms, or for their irregular development, in view of the comprehensiveness of the lesion and its different degrees of intensity in different regions. The observation of the fact of regional variations is not limited to the comparatively few necropsies in which uncomplicated general contusion has been found to exist, but is even redundantly confirmed in the far greater number in which death has resulted from hæmorrhage, arachnitis, or extensive laceration. It is not unusual in case of a contusion which involves the entire brain to find that its structural evidences are emphasized in one hemisphere or in certain lobes or in certain regions; it may be in the cortex, the basal ganglia, or elsewhere. It is not more unusual to find in a largely diffused contusion that some part, as the cortex, one hemisphere, or the cerebellum, has practically escaped. All the characteristic structural alterations are alike subject to localization. The post-mortem inspections of the brain which I have directed have demonstrated also the instability of the parenchymatous serous exudation; this not only gravitates to dependent parts, but can often be freely expressed by the hand after section has been made. The dropsical effusion moves through the brain substance with the same certainty, if not with the same celerity, that it does through subcutaneous cellular tissue. There is no more reason to question the fluctuation during life in the amount or position of serous transudation or in the intensity of hyperæmia originally established by violence, than there is to doubt their often progressive increase or diminution. The punctate hæmorrhages into the brain substance are, of course, not subject to change, but I believe them to be less influential in the modification of symptoms than the conditions previously described.

These considerations seem sufficient to account for the wide variations noted in symptomatology. It is unnecessary to review the cases which illustrate the dependence of symptoms of cortical irritation upon cortical contusion of the vertex, or of pressure symptoms upon excessive general subcortical hyperæmia and œdema, or of various other combinations of symptoms with structural changes. It is quite possible that wider observation may further illumine the invasion and march of symptoms, but as these must continue to depend upon unstable conditions they are not likely even then to become fixed elements in diagnosis.

(2) LIMITED CONTUSION.—The distinctly limited form of contusion as distinguished from laceration demands but brief consideration. In the occasional instances in which it occurs in scattered areas through the centrum ovale it can afford no indications separable from those of a modified general lesion. In its more usual form, in which it is confined to the cortex, it differs from laceration only in the extent of local injury to tissue, and the character of the symptoms will not be further influenced by the fact that the injury is a bruise rather than a wound. It is rarely a fatal lesion, and its existence is likely to be marked by the coexistence of others of greater magnitude or severity. It has been noted in but fifteen of the necropsic examinations which I have made, and in none of these had it appreciably contributed to the fatal result, and in but one occasioned recognizable symptoms. In the exceptional instance there had been no reason during life to suspect that there was a limited contusion rather than laceration.

(3) LACERATION.—I have expressed a doubt whether laceration of the brain occurs without some degree of contusion. I may add that a resultant cortical hæmorrhage, usually proportionate to the extent of local injury, and often sufficiently large to have an intrinsic value in the development of symp-

toms, is almost certain to exist as a complicating condition. I have failed to recognize one or both of these attendant lesions in but few instances, and from the time I began to record the full results of necropsic inspection, the accessory lesions as well as those which I regarded as essential, I have found the rule to be practically absolute. It is probable, however, when post-mortem indications of general injury are not pronounced, and laceration is extensive, with no more than moderate cortical hæmorrhage, that the significant symptoms have been derived from the local destruction of tissue.

I have collated ten cases in which laceration has been considerable, and in which cortical hæmorrhage and general contusion have been apparently insufficient to be symptomatically important. They include both cortical and subcortical injuries, variously situated upon and beneath the several surfaces of the brain, and have involved both localizing and non-localizing areas.

Consciousness was ordinarily lost in the beginning, though in two instances there was simple obscuration of the mental faculties, and in one consciousness was retained and the mental condition was unimpaired. Delirium very generally followed, often characterized by restlessness rather than by violence, or accompanied by fixed delusions. In one rather lengthened case there was a single delusion in which the patient never faltered, even at times when his intellectual poise was otherwise undisturbed. The subsequent progress of the case, when death was not an early termination, was likely to be marked by evidences of mental decadence. Irritability, convulsions, loss of fæcal and urinary control, were not infrequent symptoms. The pupillary condition was variable, as previously noted in hæmorrhages, and, with the characters of the pulse and respiration, will be made the subject of later consideration.

The examination of temperatures is quite as instructive as in any of the conditions previously studied. In two instances the temperature was not recorded; in the eight cases remaining the record is worthy of reproduction in brief:

*Case VI.*—103° on admission; 102° in five hours, and progressive rise to 106·2° at death in twenty-four hours.

*Case XX.*—101° on admission; 104·8° in forty-eight hours and for seventy-eight hours afterward; 101°+ to 102°+ for next ensuing forty-eight hours; and 107°+ at death in seven days seven hours. Rise progressive.

*Case XXI.*—104·8° on admission, after eighteen hours, and death in twenty-six hours.

*Case XLIV.*—98° on admission; 103·6°; 104·6°; 106·6° half hour ante mortem; death in nine hours and a half.

*Case LII.*—98·6° on admission; 104·7° in five hours; 103·8° to 103° on second and third days; 101° to 99° on fourth and fifth days; 99·8° to 101·8° till the end of the twentieth day; 102·4° on the twenty-first day; 105° on the twenty-second day; 97·5° before death on the twenty-third day.

*Case LIX.*—99·8° on the first day; 104·6° on the second day; 103·6° on the third day; 103·2° on the fourth day; 107° to 108·2° on the fifth day. Death.

*Case LXXIII.*—99·2° on admission after twelve hours; 103·8°; 104° *in articulo mortis;* 106° half hour post mortem. Death in ten hours.

*Case C.*—100° on admission, thirtieth day; 104°+ to 102° from thirty-first to thirty-fourth day; progressive rise to 107·6° at death after operation on thirty-sixth day.

These temperatures call for little comment. Their remarkable primary elevation, following recovery from shock, and in early fatal cases their rapid and progressive increase, sometimes continued even after death, with in general only brief and unimportant recessions, are in such contrast to what has been observed in hæmorrhages, meningeal inflammations, or even general contusions, that they are sufficiently striking phenomena to at once challenge and arrest attention. In a single instance in which in the last hours of life the temperature became subnormal, the exceptional fact is probably explicable by the asthenic condition finally induced by an advanced necrotic process.

A more comprehensive, and at the same time more accurate, conception of the symptoms due to laceration may be obtained from a review of the much larger number of cases in which limited destructive lesions are attended by other anatomical changes, perhaps equally important. It is only necessary to exclude those symptoms which have been found to be referable to each of the attendant lesions as they have occurred elsewhere in comparative isolation.

I have subjected to analysis forty-two cases of this type, in all of which the laceration has been distinctly marked, and in most of which the history and necropsic record have been fairly complete. An occasional case has been admitted to consideration in which historical detail has been lacking, but in which some characteristic symptom has been strikingly displayed. The region of the brain involved or the nature of the complication has not been regarded.

The primary loss of consciousness which has been observed to precede the development of positive symptoms in the history of each variety of encephalic injury, and of each combination in which they have been heretofore presented, could hardly fail to characterize this series of complicated cases in which no new form of structural alteration exists. Variations in its degree or persistence, which have been noted in connection with other lesions, equally occur in the train of lacerations whether simple or complicated.

The negative phenomena, if such a term is permissible, are indistinguishable if not identical, whatever lesion or lesions may be afterward discovered. This is readily comprehensible upon the assumption already made in case of hæmorrhages, that some degree of general contusion attends all other intracranial injuries, and that to it, in conjunction with possible general shock, the immediate effect of traumatism is to be referred. If life is prolonged, the primary morbid condition will be replaced or supplemented after a variable period by the characteristic symptoms of the coexistent lesions. In cases in which consciousness has been retained from the first, as occasionally happens, the mental condition is often peculiar; it is not that of partial consciousness or of stupor, but rather of blunted perception. The patient seems lethargic, and, if sufficiently roused, apparently comprehends simple questions in a dull way and with effort; but the effort is quite likely to fall short of his making answer; he feels and sees, but scarcely thinks. From this condition he may immediately pass through somnolence or complete unconsciousness into coma and death, or he may at once regain his mental equilibrium. In a considerable number of instances, in place of this direct solution of a psychical problem, a new series of mental phenomena are interposed between the primary unconsciousness, or the condition of lethargy to which I have referred, and ultimate recovery or death.

In the previous paper upon injuries of the head I sketched in outline certain manifestations of mental disorder which I attributed to the general class. I am convinced from further observation and more careful analysis that their significance in symptomatology should have been restricted to injuries of the parenchyma, and mainly to such of them as were of the limited and destructive variety. I spoke of these mental aberrations as of no more than "rather frequent" occurrence, but when restricted to their proper place, as indicative of the lesions which they really represent, they become more nearly pathognomonic; they exist in some degree, or in some combination, in a very large majority of the cases which survive the initial stage. I have seen no reason to materially modify the picture I then presented. In a typical case, delirium of some grade or character follows or precedes restoration to consciousness; it may be violent and simulate the alcoholic form of mania, but oftener the patient is simply restless, excitable, incoherent, or perhaps inarticulate in speech, his mind distracted by fleeting fancies, yet amenable to control. A little later he may recognize his friends, converse intelligently and coherently, and during the day and upon cursory examination appear quite rational, though still delirious and requiring mechanical restraint at night. He has delusions, fixed or transitory, and his memory is defective or entirely wanting in regard to circumstances or occurrences which preceded his restoration to consciousness. He has perhaps no knowledge of his place of residence, occupation, or family; but whatever else he may remember or forget, he is usually absolutely oblivious of all the circumstances attending his injury, and has no apprehension of his present surroundings. His nocturnal delirium may soon disappear, and eventually, after the lapse of weeks or even months, his mind may become clear, his memory be restored, and his recovery complete. In a certain proportion of similar cases the termination is less fortunate, and some degree of permanent dementia remains. In many others, unhappily, the mental horizon never brightens after the inception of delirium, or, if at all, for a brief time only, and death is not long delayed. In another type of mental disorder a condition of apathy or hebetude follows active delirium, and is likely to be merged in final unconsciousness.

In still other cases delirium is of a muttering character from the beginning, or from an early stage, and is accompanied by stupor. The occasional instances in which delirium, like absolute retention of consciousness, occurs as a primary condition probably concern the complication, general contusion, rather than the laceration.

It may be worth while to call attention to the frequent want of correspondence observed between the severity or mildness of the invasive psychical symptoms and the final outcome of the injury. A violent commencement has not always involved an answerable sequestration, and so too a good beginning has sometimes made a very bad ending; but in either instance failure to forecast the future does not necessarily imply inability to recognize pregnant symptoms. Practically the existence of the lesion has been as legibly stamped upon the histories of such cases as upon those which have run a more conventional course.

There is a peculiar irritability or sensitiveness to external impressions which I have noted as of frequent occurrence, and have ascribed to cortical injury. It is an exaggerated response to trivial irritations and disturbances which seems due less to cutaneous or muscular hyperæsthesia than to an abnormal excitability of the emotional centers. Great vexation and impatience are often manifested from slight irritation, even in the last hours of life when the patient has remained motionless and apparently unconscious for a length of time.

Aside from symptoms connected with the perceptive and intellectual centers, loss of control over the bladder and rectum is of first importance among general indications. It is impossible to estimate its numerical frequency, for if these receptacles are empty at the time the brain injury is inflicted, and if, as often happens in recorded cases, life is afterward measured by hours or minutes even, this diagnostic point is necessarily lost. If such explicable cases are excluded, it may be said to have been very generally observed in the clinical studies of laceration which I have made, though it has sometimes failed of record. It has been equally noted in the absence of any form of paralysis, and when consciousness has been retained; and though some form of mental impairment may have always coexisted, the same loss or aberration of mental power when due to other lesions has not been characterized by this particular functional incapacity. The lacerations have been both cortical and subcortical, and have involved all the lobes and all regions of the brain, so that the direct cause of this lack of control would seem to be any wound of the parenchyma, whether or not it may be ultimately traced to some special center.

Convulsions have been of comparatively frequent occurrence in the fatal cases. I recall but one instance of subsequent recovery in which laceration was fairly inferential. In the fifty-two cases upon which thus far conclusions have been founded there were general convulsions in ten, convulsive movements in three, and general muscular rigidity independent of meningeal inflammation in two. This is largely in excess of the proportion of cases in which this symptom occurs in hæmorrhages, of which it has been supposed to be characteristic. I have never known it to follow simple general contusion even when of marked severity. I believe the pathic condition upon which convulsions depend to be distinguishable by certain peculiarities in their manifestation. In hæmorrhages they result from compression or concomitant injury of the recognized motor area. In the thirteen cases enumerated in which they followed laceration, the seat of injury was usually in the frontal or temporo-sphenoidal lobes, or in both together; in two exceptional instances, the optic thalamus was lacerated in one, a parietal lobe was wounded by a fragment of bone in an old infected compound fracture in the other. If in some cases there were additional lacerations of other lobes, they were of secondary importance and distant from motor centers. It is obvious that when convulsions result from an intracranial hæmorrhage which has relation to motor centers, they are likely to be preceded or accompanied by paralysis, as in my own cases, or in three recently reported by Dr. A. J. McCosh. If they are induced by laceration, which as it has been shown is in general frontal or temporo sphenoidal, paralysis is an unlikely factor in the case. It really occurred in but two instances: once in the compound parietal lesion mentioned, and once in a frontal necrosis surrounding a small tumor which was only an incident in a general traumatism.

The characters of the associated symptoms—of hæmorrhage on the one hand and of laceration on the other—aid much in determining the significance of a convulsion. The temperature which precedes the paroxysm has special value since it is a very early indication of the nature of the lesion and of positive character. In every instance within my observation it has been distinctly higher than that which accompanies hæmorrhage. The immediately subsequent temperature has, of course, no diagnostic importance. I have been able to discover no absolute law which governs the invasive or initial symptoms, though the first spasmodic movements are perhaps rather more frequently developed upon the opposite side.

The paralyses and anæsthesias which may follow laceration are of great assistance in fixing its location after the nature of the lesion has been established. Their very general origin, however, in hæmorrhages, inflammatory effusions, or depressed fractures does not warrant the assumption from the mere fact of their existence that there has occurred a destructive lesion of the brain substance; but the laceration having been determined by other considerations, these conditions may be quite sufficient for localization. Their connection with laceration is exemplified in many of the cases which I have described. The subject of paralysis and anæsthesia in general is sufficiently well understood to obviate the necessity of giving it any special attention in the present review of symptoms.

The irregularity of the pupils has not impressed me as of greater symptomatic importance in this than in other encephalic lesions. The forms and combinations of pupillary variation have been so numerous, and the instances in which no pupillary changes have occurred have been so frequent, that I have come to doubt the practical value of the indications which they afford. The ten cases of comparatively uncomplicated laceration, quoted heretofore, will serve to illustrate the inconstant relation which exists between the condition of the pupils and the nature of the brain injury. In four cases there was no pupillary change, though in each one laceration was extensive, and included in one instance an excavation of an entire frontal lobe, in two others cortical destruction of both parietal lobes at the vertex, and in the fourth a considerable laceration of a frontal lobe at the base, besides subsidiary injuries of the temporo-sphenoidal and occipital lobes, the cerebellum, and a corpus striatum, variously distributed. In two cases in which both pupils were very moderately dilated there was gunshot laceration of a parietal lobe in one, and a laceration of the inferior surface of both frontal lobes in the other. In two other cases there was slight dilatation of the corresponding pupil with laceration of the frontal, parietal, and occipital lobes in the first, and of the frontal and temporo-sphenoidal lobes in the second. In a case of laceration of the lateral aspect of a whole hemisphere there was dilatation of the pupil on the opposite side. In the final case there was contraction of both pupils attending similar laceration of the lateral aspect of a hemisphere, with slight injury of the cerebellum and a general cortical hæmorrhage. I am incapable of understanding how any general law is to be derived from the comparison of such data as these cases afford. The only generalization which I have been able to make is that the pupils are more frequently normal than in cases of hæmorrhage.

The high temperatures which characterized simple lacerations were maintained in the presence of complications. In the forty-two complicated cases analyzed the initial observation was made immediately upon admission, but was not recorded as primary if some hours or days had elapsed after the reception of the injury; the ultimate observation was denominated final only when made nearly or quite *in articulo mortis.* Rectal temperatures only were noted.

The primary temperature was unnoted in two cases, was normal in one, and was subnormal in six; in the remaining thirty-three it was 99° to 100° in twelve cases, 100°+ in six cases, 101°+ in ten cases, 102°+ in two cases, 103·6° in one case, 106° in one case, and 106·6° in one case.

The final temperature was 109° in one case, 108°+ in two cases, 107°+ in eight cases, 106°+ in eleven cases, 105°+ in eight cases, 104·6° in one case, and 103°+ in three cases.

In thirty cases in which the intermediate temperatures were recorded, they were in twenty progressive and without recession from the beginning to the end. In recovering cases the reduction of temperature from 99°+ to normal was very slow and often extended over several months.

These very considerable elevations of temperature have been often coincident with lesions which have involved what have been described as thermo-genetic centers, and rather noticeably that part situated about the antero-inferior aspect of the corpus striatum. It is also true that laceration of any portion of the brain will be followed by a high temperature, and that the regions in which these supposed centers are situated are most subject to injury. It is therefore questionable how far the study of traumatism has confirmed in this regard the results of certain physiological investigations.

I am still unprepared to estimate the possible importance of bilateral variations in axillary temperatures. So far as my observations have yet extended the results are uncertain and often apparently inconsistent with each other, even in the same case. Temperature is sometimes uniform upon the two sides of the body, but is oftener higher upon one, which is not always the same with reference to the side of the head upon which the lesion is situated. The differences have ranged from two tenths of a degree to, in one instance, nearly three degrees. I am inclined to believe that this variation exists more uniformly in cases of intracranial injury than under other conditions, and that the temperature is rather more frequently two tenths of a degree higher upon the side opposite, than upon that corresponding to the seat of injury. I have as yet insufficient data to form definite opinions as to its significance and pathological relations.

In eleven cases, including the one to which reference was made in the class of simple lacerations, the progressive rise of temperature was continuous for a certain time even after death; they constitute a considerable proportion of the cases in which post-mortem observation was made, and are best shown in tabular form :

| Case Number. | Final temperature. | Post-mortem temperature. |
|---|---|---|
| 25 | 104·8° | 106° |
| 30 | 106·8° | 109° |
| 33 | 100·4° | 101 2° |
| 34 | 100° | 102·6° |
| 117 | 107·4° | 109·4° |
| 60 | 108·6° | 110° |
| 61 | 107·8° | 108° |
| 125 | 107·2° | 107·8° |
| 128 | 106° | 106·2° |
| 73 | 104° | 106° |
| 130 | 105·6° | 106° |

The lesions associated with this post-mortem calescence, which extended over an hour or more, comprehended all those heretofore described, and involved all parts of the brain; the only one which was constant was laceration; but even this was sometimes disproportionate to the severity of a general hyperæmia, or to the amount of a cortical hæmorrhage, by which it was attended. I am unable to trace any connection between this phenomenon and the region of the brain affected, and it certainly has no dependence upon injury of the so-called heat centers. It seems probable that it is a mere continuation of a thermogenetic process, however excited, or the result of deficient thermollysis, however occasioned.

I shall refer to the pulse and respiration in only general terms. Their one notable characteristic was a very slight deviation from the normal standard. In the whole number of cases which I have examined, in which laceration was the essential factor and in which there was no tangible interference with the ponto-medullary region, neither the circulatory nor the respiratory function was sensibly affected until late in the progress of the case. None of the positive symptoms have been more constant in such injuries as a class than these negative conditions. If general shock was intense, as in case of some gunshot wounds or in crushing injuries of the vertex, if hæmorrhage was excessive, or if arachnitis at once supervened upon meningeal contusion, both pulse and respiration were frequent; but these cases were exceptional. The contrast habitually presented by a practically normal and unaccelerated pulse and respiration, with symptoms of perhaps great severity, seems scarcely less remarkable than the exaggerated temperatures which have been the occasion of surprise in the same series of cases. There have been comparatively few instances, either fatal or recovering, in which the pulse has exceeded ninety or the respiration twenty six or twenty eight in frequency; they have oftener ranged well below than above this rate. The pulse when not entirely normal has inclined to fullness and slowness. The transient stimulation and subsequent paralysis of the medulla, with corresponding retardation and acceleration of the pulse, which von Bergmann attributed to a hypothetical functional disturbance of nutrition, has not yet come within my observation.

I have deferred until the present time the consideration of a condition of the pulse which occurs not only in connection with laceration, but with other forms of intracranial lesion, and which I believe has been only observed in my series of cases. I refer to a lack of symmetry in the radial pulsation upon opposite sides of the body. It was first noticed in May, 1893, and since then has occurred in an aggregate of twenty cases. The bilateral variation in the character of the arterial pulse consists in a difference in its fullness and strength. In some of these instances its strength and fullness at one wrist were in startling contrast to its weakness and tenuity at the other. It was equally regular and frequent and in all other respects symmetrical upon the two sides. In each case the existence of this difference was confirmed by two or more observers, and if not indisputable was rejected as a symptom. Eleven cases terminated fatally, of which nine were subjected to necropsy. In the cases of recovery, as well as in those which were fatal but failed of necropsic inspection, the nature of the lesions was sufficiently evident from other indications. The lesions discovered in necropsy differed in each case from those in any of the others. They comprised all forms of hæmorrhage, epidural, cortical, and pial; lacerations more especially of the frontal and temporo-sphenoidal lobes, but also of the parietal lobe and of the pons, corpora striata, and optic thalamus, and almost invariably some degree of general contusion existed. There were two cases of hæmorrhage without laceration, and one of limited contusion without hæmorrhage. The inferential lesions, those occurring without opportunity for necropsic inspection, were somewhat less diversified. They included four cases of depressed fracture of the vertex, with moderate general contusion in three, and with epidural hæmorrhage and laceration of the frontal lobe in one; four cases of fractured base, with laceration of frontal and temporo-sphenoidal lobes in three, and with hæmorrhage and general contusion in one; two cases of laceration of the frontal lobe, with parietal hæmorrhage; and one case with simple general contusion.

The pulse was fuller and stronger on the side corresponding to the seat of injury in eight cases, upon the opposite side in nine, and in two this relation was unknown, from imperfect clinical record in one instance, and *ex necessitate rei*, in a case of general contusion, in the other.

It would seem impossible, therefore, to infer the character or location of the lesions from this symptom alone; it is equally so from any correlation which exists between it and others by which it has been accompanied. The first few cases seemed to indicate a suggestive connection with the pupillary condition, which larger experience has shown to be fallacious. The pupils are dilated in a considerable number of cases, normal in an almost equal number, and contracted or asymmetrical in others.

*The Extent to which Traumatic Lesions Aid in the Determination of Centers of Functional Control.*—The present study of intracranial lesions has been, up to this point, independent of their relation to special areas of functional activity. The question consecutively arises as to the possibility of connecting symptoms with the seat of injury in accordance with known laws of cerebral localization. The difficulties in obtaining clinical confirmation of the inferences derived from physiological experiment, which have been recognized in the examination of idiopathic disease, are exaggerated in case of the complicated lesions of traumatic origin. The number of cases in the series which I have collated is sufficiently large to have a certain value, either positive or negative, in determining how far such a relation exists. It is conceded that a motor zone, contiguous to the Rolandic fissure in the human brain and analogous to a similar area experimentally demonstrated by comparative physiologists, has been heretofore abundantly verified by observation of both idiopathic and traumatic lesions. This is also sufficiently illustrated in the cases which I have described, though in a relatively small proportion of their whole number, since violence, even when inflicted upon the vertex, is so generally transmitted to the base, where its limited destructive effect is exerted, that the motor region is likely to escape from injury. The general and local paralyses which follow the functional or structural impairment of the motor centers are so well understood that further reference to them as they have occurred in my own cases may be properly omitted.

A much larger proportion of the cases which I have instanced relate to injuries sustained by regions of the brain in which function has been less successfully studied in the light of clinical observation. These have presented symptoms which are to a certain extent diagnostic, and at the same time incidentally confirmatory of views of cerebral localization founded upon physiological induction.

No part of the brain has been so frequently involved in fatal injury as the frontal lobes. They have been lacerated in more than one third of all the cases which I have subjected to necropsic examination. It is evident that unconsciousness or delirium attends any form of lesion situated in any region of the brain; but mental disorder or decadence, apart from these, has been supposed to be dependent upon a definite and limited structural alteration, and assumed to be of the prefrontal convolutions. The influence of direct frontal injury in so many cases upon the integrity of thought or its manifestations can hardly fail to be of assistance in determining the accuracy of this localization.

The series of one hundred and thirty necropsies includes forty-nine instances of laceration of one or both frontal lobes. In twenty-three cases morbid mental conditions had been inappreciable through unconsciousness, which was both primary and permanent. In the remaining twenty six mental changes were observed in nineteen. An examination of the seven cases of frontal laceration which were without mental derangement shows that one, in which early symptoms were not obtainable, involved both lobes; the other seven were confined to the right side; so that, in practically every instance, every one with a history in which the left lobe was lacerated, there were evidences of mental default or aberration. The special manifestations of disordered intellect which they presented have been outlined in individual histories and scarcely require repetition in detail. Loss of memory, especially of the fact, manner, or circumstances of injury, confusion of ideas, inability of comprehension, incapacity of mental concentration, incoherence, fixed or transitory delusions, apathy, hebetude, or stupor, were of constant occurrence, singly or in combination with each other. A condition of mental confusion and incoherence with delusions, which occurs at a late period, is often confounded with the early delirium of cortical irritation. The lacerations were not always of the same character, situation, or extent. Five had led to almost complete subcortical disintegration, eight of the cortical injuries were confined to the base, and the others, wholly or in part, were upon the antero-superior surface. In one instance the interior of both lobes was practically destroyed. The symptoms held some relation to the nature and extent of the lesion; in the subcortical excavations there was in each instance abrogation of mental power, rather than an aberration in its manifestations. The patient's condition was noted at the time in the several cases as "sluggish," "apathetic," "without sign of intelligence," or as "apparently devoid of power of comprehension." It was generally characterized by torpidity and indifference. In the cortical injuries, in place of comparative default of intelligence, there was incoherence, perverted memory with delusions, or the stupor which comes from confusion of ideas and mental indifference; the mind was alert to external impressions, though they were not always rightly comprehended. It is a noticeable fact that in a large proportion of cases the superficial injuries were upon the inferior surface, which has been classed as a latent area. The distinctions which I have made in the mental condition, as it follows cortical or extensive subcortical laceration, are broadly drawn, but I believe will be found to be justified in an examination of the cases which I have cited.

The converse proposition that frontal laceration alone, of all traumatic conditions, occasions a direct loss or derangement of intellectual function, independent of delirium or unconsciousness, is only a little less absolutely true. In the same series of one hundred and thirty necropsies death had been preceded by such deficiency or derangement in four instances in which this injury was not disclosed. In one of these, a case of gunshot wound of a parietal lobe, some slowness of comprehension was the only mental symptom aside from a hysterical melancholia which had led to a suicidal attempt; this may be properly excluded, as mental disease existed before the reception of injury. In each of the other three mental disorder and subsequent decadence were well pronounced. In one, general hyperæmia and œdema were excessive, with a small hæmorrhage into the substance of an optic thalamus; in another, simple general contusion with œdema only existed; and in the third, a large localized subarachnoid serous effusion compressed the frontal lobes. The exceptional cases, but three in number, in which these lobes, though not lacerated, were still the part solely affected by a limited lesion in one and included in the general lesion in the other two, can not be said to controvert the presumptive evidence derived from physiological observation and so generally supported by the results of pathological investigation that the control of the intellectual faculties is located in this region of the brain.

The difference in the morbid mental conditions which have followed laceration, as it has affected the left frontal lobe or the right, I believe has not been suggested by physiological experiment or noted in previous observations of traumatic lesions. In every case of the present series in which consciousness was retained or regained, and in which the history was known, laceration of the left frontal lobe has been attended by intellectual aberration apart from simple delirium; in similar cases in which the right lobe has been lacerated without destructive injury of the left, there have been in their larger proportion no symptoms of mental disorder, and in the remainder there has been only stupor or active delirium, as may happen in diffused lesions and in other parts of the brain.

The examination of lacerations which involve the temporo-sphenoidal lobes has had scarcely more than a negative importance. They are forty-five in number and were attended by an aphasic condition in but a single instance. In the greater number of cases in which laceration existed, twenty eight, entire unconsciousness, the grave of so many possible symptoms, precluded its recognition. In the seventeen cases remaining it was confined to the base, a supposed latent area in eight, was unplaced in one, was an extensive subcortical excavation in three, and in four was situate in the lateral region, which includes the centers of speech. The instance of aphasia is detailed in the historical abstracts, and the essential lesion, which was purely lacerative, extended quite through the cortex of the first and second left temporal convolutions. The arachnoid was unruptured, and there was no cortical or other local hæmorrhage; Broca's convolution was unaffected. In the other cases of lateral laceration there was slight injury of the first or second convolution. None of the forty-five cases, save the one mentioned, presented any symptom, unless it were a convulsion, which could be considered indicative of temporo-sphenoidal injury. In the sixty-two fatal lacerations which comprise all those in which the frontal and temporal lobes were implicated, separately or together, there was but the one instance of aphasia mentioned. In the recovering cases it will be found to have been of more frequent occurrence.

It has been assumed that hæmorrhage compressing the centers of speech is a cause of aphasia. I am compelled to dissent from this proposition, not only as contrary to the results of my own observation, but from general anatomical and pathological considerations. The compression, if exerted by a small amount of blood, must be direct and accurately applied; if it be by a hæmorrhage large enough to include these small spaces in the wide expanse of cerebral surface through which we are brought in touch with the world without, the individual default is lost in the general obscuration of all the faculties which attends the grosser injury. A pial hæmorrhage from meningeal contusion in this region is likely to be scant and diffused; a cortical hæmorrhage, if small and confined to either area in which the control of speech resides, is derived from laceration of the part itself to which as the primary and more potent lesion the result must be attributed; an epidural hæmorrhage while yet in moderate amount acts indirectly and inadequately upon the temporal or lower frontal region through the dura which acts as an efficient shield. In wounds of the middle meningeal artery, in which the effusion of blood may in time become excessive, the loss of consciousness which then ensues abrogates speech with all the other manifestations of intellectual life, and there no longer remains a question of aphasia.

I have never met with a pial or cortical hæmorrhage of local origin which suggested an interference with the integrity of speech, nor one where consciousness had been retained or restored in which blood had descended from the vertex in amount sufficient to produce this result by compression of the frontal or temporal lobe. My experience has assured me that such an event, if it happens at all, is much too unusual to justify the statement made that it is a contingency to be expected. I have seen cases of large epidural hæmorrhage in which consciousness was gradually lost before death or relief by operation, but I have never recognized aphasic symptoms at any time during their progress.

There is a case of motor aphasia attributed to hæmorrhage reported by Dr. M. A. Starr in which the patient was trephined with great benefit. The amount of blood was small and limited to the motor region. He immediately regained some power of speech, and a little later indicated some mental improvement. I have already expressed a belief that intellectual and emotional impairment is not occasioned by traumatic hæmorrhage. There were evidences of both in this case which the amount of blood discovered and removed was certainly insufficient to explain. It is necessary to assume laceration in order to account for their existence, and it seems more than probable that the same lesion occasioned the aphasia; it might readily have escaped notice in the comparatively small opening of operation, more especially if it were entirely subcortical within the visual area. The patient, I am told, after a lapse of years is still aphasic, a fact difficult to understand if dependent upon so small a hæmorrhage as described.

It by no means follows that hæmorrhage was the cause of symptoms because immediate improvement followed operation. The removal of a small portion of bone not infrequently relieves morbid cerebral conditions though the lesion remains undiscovered and unknown. Examples of successful results from operative failure in cranial surgery are as varied as the conditions which demand interference; there is one such in the present series of cases * in which traumatic convulsions of several days' continuance were im-

---

* No. 120, original series.

mediately and permanently controlled by trephining both in the region of direct injury and at the supposed point of *contre-coup*, though nothing abnormal was discovered and nothing more was done.

I am unable to accept this case of aphasia as a result of hæmorrhage even as an exceptional phenomenon.

There are a number of instances of laceration of the cerebellum, but they can be hardly said to have afforded distinctive symptoms. It was in each complicated by other lesions, and the indications were those of laceration and hæmorrhage in general, and it might be with added localizing signals of injury to areas of which the function has been more accurately defined. If the cerebellum has any concern in the maintenance of bodily equilibrium, it is not likely to be disclosed in traumatic cases.

The pons was occasionally contused; it was hyperæmic, œdematous, or in some part studded with hæmorrhagic extravasations, varying from the size of a robin shot to an effusion a half by a quarter of an inch in its diameters; but resultant symptoms, if they existed, were merged in the general traumatism.

In some cases of injury of the medulla there were respiratory changes of importance, perhaps it might be better said, of interest, since they preceded death by so short an interval that there was no longer question of prognosis or of treatment. The constant lesion was some form of hæmorrhage causing direct compression. An extension of a hyperæmia from the pons, or even of a slight œdema, seemed to be void of effect. The symptoms were those of pulmonary œdema, cyanosis, or a marked reduction in the frequency of the respiration. The first and second might be considered characteristic after exclusion of other causes of apnœa; the infrequency of respiration when progressive and extreme is almost if not quite pathognomonic. In three instances in which the respiratory acts were reduced to no more than two in the minute, radial pulsation was continued for two and three minutes after respiration had entirely ceased, as sometimes happens after mechanical occlusion of the larynx or trachea.

The more inaccessible regions of the brain are not exempt from destructive alteration. I have described cases in which the corpus striatum, the optic thalamus, the corpus callosum, the gyrus fornicatus, or a portion of the fornix was contused or lacerated, and this was sometimes the only localized injury in the midst of general contusion. There is the same insuperable difficulty in connecting symptoms with lesions of the optic thalami or corpora striata as existed in case of the cerebellum. They are not sensitive to minor injuries, and in the severer lacerations, which I have seen extend even to practical disintegration, the patient may lie motionless and unconscious with no apparent symptoms which are not afterward explained by discovered lesions of the adjacent motor or sensory areas.

I have unsuccessfully endeavored to verify the existence of heat centers in the human brain by an examination of temperatures following intracranial traumatism, though such centers seem to have been experimentally demonstrated in the lower animals. The multiplicity of lesions, their wide extent and indefinite outline, render the results of accidental injury necessarily uncertain in comparison with the accurately limited cerebral wounds which are inflicted in vivisection. In some instances very high temperatures have attended laceration of regions which correspond to the heat centers determined by experimentation. I have at times thought that this was especially true of injuries of the frontal lobe immediately anterior to the corpus striatum, but these were always included in more extended cortical ruptures or subcortical excavation, and I have observed that large lacerations elsewhere, even in parts unsuspected of special influence upon the control or production of heat, may be accompanied by temperatures equally exaggerated. The only limited injury of the corpus striatum attended by any considerable rise in temperature was small, and was complicated by a frontal laceration quite sufficient in itself to account for the thermic condition. In another case in which there was a large extravasation into the corpus striatum, three eighths by a quarter inch in its diameters, temperature did not exceed 101°. In still another case the right corpus striatum was completely disintegrated and the left corpus striatum and the right optic thalamus lacerated, yet the temperature rose only to 102·2° in the eight hours and a half which preceded death. In an instance of extravasation into an optic thalamus with excessive general hyperæmia and œdema, in which the clot was as large as a cherry pit, though the final temperature was 105°+, there was no elevation above 100° till the fifteenth day. Lacerations of the fornix and corpus callosum were followed by much higher temperatures. Laceration of the pons in one case developed an excessively high temperature, but in others there was no unusual elevation. Cortical centers, as they have been described, are topographically too indefinite to afford data for accurate comparison of cases. I believe, however, that the highest temperatures which I have recorded have not corresponded to injuries of tissue in what I understand to be their situation. Lacerations of any part of the cortex have been attended by marked elevations of temperature, quite as great when at the base as when in the uncertain neighborhood of the "cruciate centers."

The opinion which I have formed from an analysis of my first series of cases that high elevations of temperature from traumatic laceration of the brain were dependent upon general nutritive changes rather than upon lesion of limited thermogenetic or thermotaxic centers has not been disproved or modified by subsequent experience. It is impossible to predict the result of further clinical observation, but Dr. C. L. Dana's examination of intracranial hæmorrhages of idiopathic origin, in which he reaches the same conclusion, tends to confirm my belief in the correctness of my original impression.

I have been equally unable to connect the occurrence of certain circulatory phenomena with lesion of definitely limited vaso-motor centers of control. The lack of symmetry in the characters of arterial pulsation upon opposite sides of the body, which has been frequent, and the dark flush upon the face which I have noted in two cases of very different import, are to me as yet ætiologically and pathologically inexplicable.

The results of a general analysis of all the cases which I have recorded up to the present time, so far as they relate to functional localization, may be summarized in the following propositions:

1. That the control of the intellectual faculties resides in the frontal lobes, perhaps exclusively in the left, and that manifestations of their aberration or default are due to a destructive alteration which is almost invariably laceration.

2. That the control of the faculty of speech, resident in the frontal and temporal lobes, is impaired by structural alteration alone.

3. That characteristic disturbances of respiration are caused only by compression of the medulla from hæmorrhage.

4. That high elevations of temperature, while dependent in occasional instances upon diffused contusion of the

brain substance, are ordinarily the result of limited lesions which are confined to no special regions or centers of control.

(4) PYOGENIC PARENCHYMATOUS INFLAMMATION.—There remains a parenchymatous inflammation of pyogenic character. It is an infrequent result of traumatism and is of limited form except when produced by the intrusion of a foreign body, as of a bullet or drainage tube. I exclude cases of infection from direct laceration which sometimes follow neglected compound fracture with wound of the dura. They are surgically unpardonable, and at the present time ordinarily due to the stupidity of the laity rather than to the carelessness of the surgeon. They afford no question of diagnosis, since the pyogenic process alike involves the brain, the membranes, and the surface of the wound, and is open to visual inspection. The few cases which have been admitted to my service were recognized without difficulty.

The infrequency of central or true abscess as a result of traumatism is confirmed by my experience, in which it has occurred but twice in a series of three hundred cases—once in the fronto-parietal and once in the parieto-occipital region beneath the angular gyrus; in both it succeeded a compound depressed fracture of the vertex without wound of the dura, and was situated at an appreciable distance from the point of cranial injury. In the first case, after elevation of the bone and some primary rise in temperature, there were no general symptoms till the twentieth day, when a few drops of pus escaped from the wound and a cavity was discovered two inches and more away from the opening in the skull. At this time there was a little mental dullness and slight facial paralysis with a scarcely noticeable elevation of temperature; recovery soon followed. In the second case the wound healed, and there were few indications of trouble beyond those dependent upon some enlargement of the cervical glands till the twenty-second day, when the patient somewhat suddenly began to suffer from acute frontal headache, became delirious, somnolent, hemiplegic, and within twenty-four hours hemianæsthetic, with unconscious and involuntary evacuations, and with infrequent pulse and absolutely normal temperature. The abscess, which was small, was discovered and drained, but death ensued sixteen hours after operation. Both cases conform to the dictum of von Bergmann that no traumatic abscess occurs without wound of the integument, and are not inconsistent with the bacteriological opinion that parenchymatous inflammation of the brain is always septic. Von Bergmann, however, is in error in his general proposition. In a circumstantial report of the second of the cases mentioned, I referred to a specimen in the collection of Dr. H. M. Biggs which in connection with its history affords conclusive evidence that an "open wound in the head or soft walls of the cranium" is not essential to the formation of a traumatic abscess. In this case a youth of seventeen was struck in the forehead by a baseball and suffered temporary unconsciousness; there was neither cranial fracture nor wound of the scalp, and in two or three days he had apparently recovered, though headache persisted. Later there was impairment of vision and eventually total blindness. His death occurred suddenly about six months after the reception of the injury. The abscess occupied the whole left frontal lobe, encroaching upon the parietal, displaced the tentorium backward, and compressed the right hemisphere. It is unnecessary to multiply instances, as a single one is sufficient to show that a possible general rule is not to be regarded as absolute. The distinction is important in diagnosis, for in a large class of cases it prevents the exclusion of a condition which always must be reckoned with as a possibility at least. I have no doubt that the external cephalic wound usually exists, and that as the shortest route by which the pyogenic germ can travel it is the one by which entrance to the brain is likely to be expected.

In the report to which I have referred I attributed the location of the abscess to a limited subcortical contusion and, as I think, demonstrated the correctness of my conclusion. Von Bergmann again errs in making a statement too positive when he denies the existence of this lesion except as a product of extreme violence, which "shatters the entire skull." Bruises and lacerated wounds of the brain are demonstrably very much more frequently superficial than subcortical, but instances both of laceration and of areas of limited contusion in its deeper structure, when no extraordinary violence has been inflicted, have been often enough disclosed in this series of cases to make it evident that if infrequent they are hardly to be considered unusual. It is rather the surface abscess, which he believes to exemplify the rule, than the central contusion and abscess which is to be considered exceptional. Pott's puffy tumor, the surface abscess, and even hernia cerebri as a factor of importance, have practically disappeared from the field of American if not of German surgery. In fact, any form of traumatic brain abscess is so exceptional that it seems idle to speak of either rules or exceptions.

The immediate cause of the pyogenic process is a question which so exclusively concerns the bacteriologist that I shall not venture to enter upon its discussion. Dr. Biggs, who commands my confidence, regards all pyogenic processes in the brain substance as of septic origin, and since the colon bacillus has been found in the product of idiopathic arachnoid inflammation I am quite prepared to believe that the streptococcus or any other germ can in some way reach the nidus formed by the bruising of the parenchyma, even though a direct route has not been opened through a cranial wound.

It is, of course, impossible to discuss symptomatology or diagnosis upon a basis of observation derived from two cases, and this study of intracranial lesions in their traumatic aspect has been confined to the results of personal experience. I call attention only to the possible relation of acute posterior cervical glandular enlargement, which occurred in the second case, to the beginning of cerebral inflammation.

The term encephalitis has been rather freely used to express a supposed result of intracranial injury. I believe that in the proper sense of the word no such condition exists. In case of any infected cranial wound pyogenic inflammation may extend through the meninges and include the cortex, or a diffuse purulent inflammation of a portion of the parenchyma may even reach the meninges, and in any acute arachnitis the contiguous brain surface may be infiltrated with cells; but anything like a concurrent general inflammation of all the cranial contents is unknown to me either from observation or from definite record. It is often held to be synonymous with cerebritis, which as a general parenchymatous inflammation, independent of the pyogenic process which I have mentioned, I regard as no less apocryphal. I have procured the minute structural examination of many hyperæmic and œdematous brains taken from patients who had survived injury for some days or weeks, but in no instance has any evidence of the inflammatory state been discovered. This result is in accordance with the opinion of Dr. Biggs which I have already quoted. I have read numerous vague descriptions of traumatic encephalitis or cerebritis, but none in which the disease was fitted to the name, or in which it was connected with precise pathological changes. I am too well aware of the danger of unqualified statements to deny absolutely that it ever exists; but, so far as I am enabled to judge, it is no more than a misapplication of words or pure assumption.

### CASES NOT VERIFIED BY NECROPSY.

It seems proper at the conclusion of a summary of cases in which the value of symptoms has been established by necropsy to refer to those which by reason of recovery have been heretofore disregarded as incapable of affording positive evidence of the pathogenic condition upon which they depended. I believe no differences in symptomatology will be found to exist except in degree, and in many instances the early progress of the case is not at all indicative of the final result. There is probably no symptom which occurs in fatal cases which may not be noted in those destined to a more favorable termination, except the infrequency of respiration which follows compression of the medulla; and no other symptom which is less characteristically present, except elevation of temperature. Even in temperature the distinction is not absolute; it does not apply in cases of hæmorrhage, and only to the later stage of meningeal inflammations and of the morbid conditions occasioned by lesions of the parenchyma. Not only in the beginning but for a considerable time afterward temperature may rise absolutely higher in a recovering case than at any time in the course of one which is to end in death; but in general it is less pronounced. I have never known it to exceed 105°, and rarely to attain so great an elevation. The symptoms altogether exhibit no greater differences, as the issue varies, than obtain in other types of disease.

In illustration of the cases in which the significance of symptoms has not been demonstrated by the direct inspection of pathogenic lesions I have abstracted the histories of a certain number, of which some ended in recovery and others in death, but without necropsy. This class includes one hundred and four as yet unpublished cases, in addition to the sixty-six which appeared in my first series. Many of these are fractures of the base or vertex, or uncomplicated general contusions in which symptomatology, diagnosis, and results were so simple that they have scarcely more than a statistical value. I have selected from the remainder some which have special interest on account of symptoms which made diagnosis clear, or which occurred in such combination as to make their interpretation difficult.

Case I.—Male, aged thirty-five years, fell while dancing and struck the back of his head on the ballroom floor; no loss of consciousness, and no other indication of injury till thirty minutes afterward, when the right upper eyelid began to droop. On examination five hours later, symptoms were confined to right eye and appendages; complete ptosis, external strabismus, paresis of all the ocular muscles, imperfect accommodation, and diplopia; normal pupil and retina. At the end of eighteen months there was still some weakness of the ocular muscles, but no ptosis. The right pupil was permanently dilated.

Case II.—Male, aged twenty-eight years, received a contusion of the right parietal region, and on the second day began to exhibit symptoms which were observed at the time of examination ten days later: wide dilatation of left pupil; incomplete paralysis of all the ocular muscles and of the elevator of the upper lid of the left eye; anæsthesia of the left conjunctiva and of the mucous membrane of the left nostril, with loss of smell on that side; and intense and constant pain in all the parts included in the distribution of the fifth cranial nerve on the left side. No paralysis of the facial muscles. Some numbness of the left upper extremity. No other symptoms. Ophthalmic examination by Dr. Callan disclosed some cloudiness of the fundus and enlargement of its veins in both eyes; accommodation very imperfect. Two weeks after the injury the hearing in the left ear was lost. The patient is still under observation.

Case III.—Male, aged forty years, fell from a truck and struck upon his head; partial loss of consciousness; profuse hæmorrhage from right ear; wide dilatation of left pupil; temperature, 99·8°; pulse, 92, and respiration, 23. Second day: slight delirium; mental stupor; no response to questions; temperature, 100·8°. Third day: severe general convulsion, beginning in left arm and hand; both pupils afterward widely dilated; temperature, 100·2° to 100·6°. Fourth day: similar convulsion, but less severe; temperature, 99·8° to 100°. Eighth day: mind clear, but torpid; no recollection of an accident having occurred; speech slow and somewhat aphasic; headache and continued dilatation of pupils. Twelfth day: mental condition normal; temperature, 99° +. Discharged without further symptoms on the twenty-ninth day.

Case IV.—Male, aged sixty-five years, fell thirty feet; consciousness lost, and regained twenty hours after admission to the Presbyterian Hospital; left hemiplegia; temperature, 98°, which fell in four hours to 97°; pulse, 70 to 80; normal pupils; lacerated wound of scalp. Transferred to Bellevue Hospital eighty-two hours after reception of the injury. There was then delirium with delusions; restlessness; no recognition of changed surroundings; normal pupils and respiration; no paralysis; temperature was 100·5°; pulse, 112. For ten days continued restlessness and at times delirium, with lack of urinary control; temperature, 102·6°; pulse and respiration moderately accelerated. After that time mental condition became normal, at first only during the day, and all symptoms disappeared. Seventeen months afterward his mental and physical condition was entirely restored.

Case V.—Male, aged fifty-five years, fell unconscious in the street. On admission, profound shock and entire unconsciousness; wound of scalp in right posterior parietal region; free hæmorrhage from right ear and uniform contraction of pupils. One hour later, rigidity of left arm and, to a less extent, of left leg. Consciousness restored in twenty-four hours, and a little later the pupils became normal and the mind clear. Temperature on admission, 98°, declined to 97·4°, and rose in twenty-four hours to 99·4°; pulse and respiration normal. On the third day, temperature, 99·6°, and only psychic symptoms; mental processes a little less slow than on the previous day, but memory defective. No recollection of anything which happened after leaving home in the early morning, some hours previous to the accident; memory of words and facts equally deficient. Upon questioning, the patient said that he lived at "No. 4 in the Ninth Ward"; then remembered that it was opposite a school, which he called "skull," and finally that it was in Grove Street. On the following day he had again forgotten the name of the street, and its mention awakened no remembrance; he misplaced many words, and could not be brought to recognize his errors. A week later he had much general headache, realized that his mind had been greatly confused, and was still ignorant of all that had happened since leaving his house. He was discharged on the eighteenth day, his temperature and mental condition having been normal for several days.

Case VI.—Male, aged forty-five years, thrown from a truck in collision; admitted in shock and still unconscious; pupils contracted; temperature, 97·5°; pulse, 52; respiration, 18; twelve hours later, temperature, 97·5°; pulse, 50; respiration, 12; in fourteen hours, consciousness restored; temperature, 98°. Second day, no recollection of injury, previous occupation, or married condition. Third day, ecchymosis over right mastoid process and extending upon the back of the ear, not previously apparent. Fourth day, the patient, after much questioning and trouble, was enabled to remember his residence and occupation; temperature, 102°. During the rest of the week his temperature declined and mental condition improved, though he was still irrational and at night required mechanical restraint. In the second week he was rational at times; he was capable of expressing the generalization that a man's mind is clearer by day than at night, and described correctly the manner in which he received his hurt, though he again forgot the circumstances and denied that he had said anything about it; he was irritable and forgetful, even of the outrage to which he considered himself subjected in the taking of his temperatures; he had delusions, saw imaginary persons, and heard unreal voices, made contradictory statements about the injury which he had suffered, and was much annoyed at the attempts which were made to get from him some coherent and consistent history. Early in the third week his temperature became normal, his memory and other mental faculties were restored, and he was discharged from the hospital.

CASE VII.—Male, aged forty-five years, mind impaired by alcoholic excess, fell one flight of stairs; consciousness retained, hæmatoma in left temporal region, profuse hæmorrhage from left ear, and slight epistaxis. Temperature, 98°; pulse, 90; respiration, 24. Second day, a little delirium, rigidity of both arms, and left facial paralysis, both upper and lower; temperature, 100·2°; pulse, 100; respiration, 24. Incision made through hæmatoma revealed linear fracture of left squamous portion extending into the base. In the three days following, the temperature and mental condition became normal and facial paralysis nearly disappeared. Two days later temperature rose to 100·5°, facial paralysis increased, left side of face and neck became swollen, and delirium supervened. From this time there were recurrent maniacal attacks, lasting less than twenty-four hours, in one of which he was transferred to Bellevue Hospital and soon afterward escaped. He was at a subsequent period sent to an asylum for the insane, and is now, after sixteen months, at home, but of recognized unsound mind.

CASE VIII.—Male, aged thirty-eight years, fell from a second story window; unconscious and delirious on immediate admission, and in same condition when transferred from alcoholic ward to surgical service next day. Compound depressed fracture of left temporal bone, extending into the occiput between the curved lines; irregular dilatation of the pupils; internal strabismus of left eye which was afterward found to be congenital; pulse slow and full; breathing stertorous; no control of urine and fæces. The bone was elevated, and an epidural clot, which extended only toward the base, was removed as far as practicable; no discoverable dural or subdural lesion. Third day, left radial pulse fuller and stronger than the right; partial left lower facial paralysis and dysphagia. Fourth day, violent delirium. Fifth day, dysphagia and cessation of bilateral variation in radial pulse; delirium continued, with a short interval in which it intermitted. The symptoms were variable until the end of the fourth week; delirium of different grades at different times alternated with periods of quietude and rational intelligence; various delusions were more or less persistent; dysphagia, lack of urinary and fæcal control, and facial paralysis still continued. After this time mental improvement was progressive, and in the seventh week the mind was entirely clear. At the end of eight weeks recovery was complete, and twenty pounds lost in weight had been regained. The temperature on admission was 97·6°, on the second day 102°, and after operation 104 4°; it subsequently declined to normal at the end of the second week, and afterward varied from normal to 99°+. The pulse and respiration were varied, but never frequent. At the end of fifteen months he suffers no mental impairment.

Case IX.—Male, aged thirty-one years; fell two stories upon an iron beam; unconscious and delirious on immediate admission; wound above the left eye and contusion of the left shoulder; loss of urinary and fæcal control, which was not regained. Subsequently the patient was usually delirious at night and stupid during the day, and without other general symptoms; he was only once or twice able to make coherent reply to a question asked. He died in profound coma at the end of twelve days. Temperature on admission was 99°, rose gradually to 105° on the ninth day, and was 104·8° just before death. The pulse was 82 on admission and the respiration 24, and both afterward varied each day from moderate to extreme frequency.

Case X.—Male, aged thirty-one years; was struck by a brick which had fallen five stories; no other immediate general symptom but unconsciousness. A compound depressed fracture crossed the median line at the vertex. Second day: there was accurate memory of events up to time of injury, no recollection of anything that occurred afterward. Third day: fragments of depressed bone were removed, leaving an opening in the skull two inches by an inch and a half in its diameters; no lesion of dura or of the sinus. Temperature on admission 104·4°, at time of operation 100°, subsequently 99°+. Pulse and respiration at all times normal.

CASE XI.—Male, aged thirty-two years; fell from his truck and struck the pavement upon the back of his head; partial loss of consciousness and delirium, which continued for three days. Fourth day: limited power of comprehension, no response to questions asked, attention fixed only with difficulty, occipital headache which was not increased by pressure or percussion, and somnolence. At the end of four weeks the patient sat up, but walked with difficulty on account of imperfect muscular co-ordination in both legs; patellar reflexes normal; mind clear but slow in action, which he himself noted; vertigo, which was not of previous occurrence; occipital headache relieved. Dr. P. A. Callan discovered upon ocular examination a neuritis, more advanced upon the right side than upon the left, and a paralysis of the ocular muscles. Temperature on admission was 99°, rose to 100·4° on the same day, was from 99° to 101° till the sixth day, 98·5° to 99°+ till the twenty-first day, and afterward continuously normal. The pulse was normal. The respiration was 12 for three days, 16 to 18 for six days, 8 to 12 for thirteen days, and afterward 16 to 20.

CASE XII.—Male, aged thirty-five years; fell one story; brief unconsciousness followed at once by delirium; extensive lacerated wound in left parietal region; hæmorrhage from left ear caused by wounds of external meatus. Temperature on admission, 101·5°; pulse, 80; respiration, 18. Delirium continued three weeks, gradually diminishing in degree and constancy; no subsequent recollection of the manner of injury. Loss of urinary control lasted one week; no headache at any time, and no later symptoms. Patient recognized his family and surroundings after three or four days.

CASE XIII.—Female, aged five years; struck by a falling box which seemed to have crushed her head laterally against the floor. Still unconscious at time of admission, but very sensitive to external irritations; slight twitching of right side of the face, slight epistaxis, slightly accelerated respiration, slow and irregular pulse; temperature, 95°; pupils sometimes normal, sometimes widely dilated, with conjugate deviation which was sometimes upward and sometimes to the left; vomiting soon after reception of the injury; hæmatoma over entire vertex, and contusion of both eyes. Incision disclosed fissures on either side of the calvarium; one extended from the left temporal fossa posteriorly across the vertex to the right occipital region, and anteriorly into the anterior fossa; another, apparently beginning in the right anterior fossa, crossed the right parietal bone and terminated in the first. The bone was depressed posteriorly and the fissure open; after elevation and removal of some small fragments considerable epidural hæmorrhage was apparent. Consciousness was fully restored within twenty-four hours, and was marked by restlessness and delirium, which continued for two or three days, after which the mental condition was normal. On the fifth day paraplegia occurred, which was almost complete from the first, and absolute on the next day, with partial anæsthesia; no paralysis of the bladder or rectum. The paraplegic condition began to improve at the end of a week's time, but very slowly; a few steps could be taken without assistance six weeks later. The temperature soon after admission rose from 95° to 98·5°, on the next day to 100·2°, and after the third day varied from 98·4° to 99·8°; usually normal in the morning. The respiration was accelerated for the first ten days, and the pulse frequent for three days.

Case XIV.—Female, aged thirty years; was thrown from a wagon while driving, striking the back of her head upon an asphalt pavement; shock, loss of consciousness for twenty minutes, and severe vomiting, which persisted during the day; temperature, 100°; not taken afterward; hæmatoma in right occipital region, and ecchymosis behind the right ear, followed by severe localized pain in the right side of the head posteriorly. The later symptoms were a muffled feeling in the right ear, with diminished hearing and blunted perceptions of taste and smell which had been noted from the time of the accident. The disorders of hearing did not continue after the fourth week, but the senses of taste and smell have been permanently impaired.

Case XV.—Male, aged thirty-three years, struck on the head with a hammer and was momentarily unconscious, after which he walked to the hospital. Compound depressed fracture of the mid-vertex; both pupils dilated; left radial pulse markedly fuller and stronger than the right till after operation, five days later; no other general symptoms. Depressed fragments of bone were removed, leaving an opening in the skull an inch and a half by one inch in its diameters; hæmorrhage from a large wound of the longitudinal sinus controlled by gauze packing. Pulse and respiration became normal on the following day, and radial pulsations symmetrical on the third day. Elevation of temperature was maintained by a slough and inflammatory conditions produced by an accidental burn. Temperature on admission was 99·2°, rose in a few hours to 101·4°, and after the operation to 102°+; pulse and respiration, normal at first, were subsequently only moderately accelerated.

CASE XVI.—Male, aged thirty years; fell twenty-five feet from a ship's deck to a raft alongside; consciousness lost for a few moments only; hæmatoma over right posterior parietal region; moderate contraction of the left pupil; right radial pulse fuller than the left; urine retained; complete paralysis of left lower extremity; nearly complete paralysis of the left arm; partial paralysis of the right upper extremity; anæsthesia of the right side of the body below the third rib; hyperæsthesia of the left lower extremity; great pain and tenderness in cervico-dorsal region, and evident fracture of the first dorsal spine; mental condition apparently normal.

During the first week vomiting occurred at least once in each twenty-four hours, and pain in the frontal and in the upper dorsal region was constant and severe. The bilateral variation of the pulse was distinct till the fifth day. The paresis, hyperæsthesia, and anæsthesia, and the contraction of the left pupil persisted in greater or less degree for several months, and a paresis of the left lower extremity and the anæsthetic and hyperæsthetic conditions and the contracted pupil existed at the time of final discharge from the hospital.

An ophthalmic examination was made by Dr. Callan, and repeated at a later period, with negative result. The eye was retracted and a little less sensitive than the other, but there was no retinal change, and no loss of power in the ocular muscles.

There was no mental disturbance till the occurrence of nocturnal delirium and restlessness at the beginning of the third week. A few days later the nocturnal delirium ceased, but the restlessness at night increased, and delusions of a painful character began to occur, which occasioned the patient much distress. The first trouble which came to him was the fancied death of his wife, and when, a little later, he became convinced that this bereavement was imaginary, he was equally positive that another delusion, the death of his child, was real, and this new conceit possessed his mind for many weeks. He suffered acute mental anguish in each instance, which could have been scarce exceeded had these pure fancies been actual facts. The facial expression grew a little stupid, and an inclination to weep was manifested on ordinary occasions, equally when the amount of cutaneous hyperæsthesia was tested, or when discourse turned upon his family afflictions, but speech was always coherent. At the end of the second month there was some improvement; the facial expression brightened, delusions were less constant and of a more trivial character, and the mental condition was less uniformly clouded. In the third month delusions altogether disappeared, and mental processes, though slow, were no longer distorted; he was enabled for the first time to recall the manner of his injury: vertigo, which had been an early symptom, still persisted.

The temperature on admission was 98·4°, rose during the day to 101·8°, and on the fifth day reached 105°. It was habitually high till late in the second month at some time in each twenty-four hours, not less than 101°+ to 102°+, the diurnal variations being also considerable. The left axillary temperature was markedly higher than the right, usually five tenths of a degree or even more. The pulse was ordinarily from 80 to 90, occasionally 60 to 70, and rarely exceeded 100. The respiration during the first month was not often less than 30 and later ranged from 28 to 24.

The patient left the hospital seven months after admission. There was then no trace remaining of the cerebral injury beyond a little heaviness of manner and a little slowness of thought. The persistence of the spinal lesion was indicated by a stationary paresis of the left lower extremity and by a continuance of the disorders of sensation which immediately followed the traumatism. The left eye was still retracted and insensitive and its pupil small.

CASE XVII.—Male, aged thirty-six years; fell ten feet from - vessel to a raft alongside and then into the water; brief period of unconsciousness, profuse hæmorrhage from left ear, slight epistaxis from left nostril, and hæmatoma in left mastoid region; single general convulsion in the ambulance followed by stertor; consciousness regained at time of admission; both pupils widely dilated; hæmorrhage from the ear recurred during the night; urine retained; temperature, 98·8°. Frontal headache continued for several days, and on the third day there was transient photophobia with contracted pupils. The bladder and rectum were controlled. Dilatation of the pupils was perceptible till the end of the second week and of the right pupil even longer. The prominent symptoms were mental; nocturnal restlessness and delirium, and a rather stupid condition during the day, were succeeded in the second week by continued delirium of a mild type with delusions. In the third week active delirium ceased, though restlessness at night persisted; the facial expression was more intelligent and speech was coherent; there was perfect recollection of the manner in which the injury had been received, and also of a similar acci dent which had occurred on the same day and aboard the same ship (Case XVI, immediately preceding), but delusions were numerous and constant. At the beginning of the fourth week the patient was restless, excitable, talkative, and had again forgotten the manner and even fact of his injury. Ophthalmic examination was made by Dr. Callan with negative result. At the end of the fourth week delusions finally ceased, and when discharged from the hospital in the eighth week there were no symptoms remaining. The sense of smell was entirely lost.

The maximum temperature was on the fourteenth day, from 101° to 102·2°; the usual temperature was 99°+ till after the fourth week, and then varied from normal to 99°. The axillary temperatures were observed from the fourth to the eighth weeks; the left was habitually, but irregularly, higher than the right. The pulse and respiration presented no notable changes.

CASE XVIII.—Female, aged seventeen years; fell from second-floor window; found in coma with profuse hæmorrhage from left ear and some hæmorrhage from the mouth; left side of face, eye, and parietal region much contused. Two hours later consciousness was partially restored and sensitiveness to external impressions recovered; hæmatemesis occurred, and at a later period, after subsidence of ecchymosis of the lids, subconjunctival hæmorrhage in the left eye was discovered; the right pupil was dilated. Six hours after admission, temperature, 97·4°; pulse, 70; respiration, 38; lack of urinary control. The hæmorrhage from the left ear continued for thirty-six hours, and was followed by a discharge of bloody serum. During the first three or four days the patient was at times noisy and restless and at times quiet. She then became rational and learned for the first time that she had met with an accident and was in a hospital; but she never knew, then or afterward, of her own recollection what had happened to her. At about the same time a protrusion of the left eye became marked, and it was discovered that vision was lost on that side. There were no additional symptoms. The temperature on the second day was 100·2°; pulse, 68; respiration, 18; and temperature afterward varied from 99° to 100°+.

At the end of the fourth week ophthalmic examination was made by Dr. Callan. The right eye was in all respects normal. The left eye was on a slightly anterior plane to that of the right; its movements were unimpaired; there was a slight remaining trace of hæmorrhage near the limbus corneæ; the pupil was moderately dilated and not responsive to direct rays of light, but acting consensually with the right; there was commencing atrophy of the optic nerve and total loss of vision. From the clinical history Dr. Callan was of opinion that a line of fracture had implicated the left optic foramen.

CASE XIX.—Male, aged forty years; found in the street in an alcoholic condition; could walk with assistance; profuse hæmorrhage from left ear; slight œdema of scalp in left occipital region; no general symptoms. The recollection of having been brought to the hospital and of previous wanderings, but not of the manner of injury, returned with sobriety. Ecchymosis of both lids of right eye appeared on the following day; vertigo and occipital headache and some pain behind the left ear existed for ten days. Temperature on admission, 98·4°; rose to 101° in the course of eighteen hours, and was afterward 99° to 100° + during the three weeks the patient remained under observation. The axillary temperatures were usually symmetrical, and when any difference was noted it was higher on the left side. The pulse more frequently exceeded 90 or 100 than is usual in similar cases. The respiration was normal.

Case XX.—Male, aged nine years; fell ten feet from a dump into a scow; consciousness lost for fifteen minutes; no external injury; temperature, 98·2°; pulse, 67; respiration, 28. Third to fifth days, right radial pulse fuller and stronger than the left; somnolence till seventh day, and recurrence on the tenth and eleventh days with a condition of mental indifference; occipital pain continued at intervals during ten days. The temperature five hours after admission was 100·2°, in twelve hours was 101°, and did not exceed that degree; it was 99° + to 100° + for fourteen days, with an occasional decline to normal for a single observation or for a few hours. The axillary temperatures were observed four times daily, and the left was habitually six tenths of a degree or more higher than the right, and sometimes the difference was as great as a degree and eight tenths; they were occasionally symmetrical, but in sixty eight observations the right was never the higher. The pulse was usually 52 to 84, and more frequently approximated the lower figure. The respiration was from 18 to 28.

Case XXI.—Male, aged fifty years; fell twelve to fifteen feet from a loft and struck upon the back of his head, six hours previous to admission; unconscious fifteen minutes; contusion of the vertex in the median line; wound in right occipito-mastoid region; hæmorrhage from right ear; delirium from time consciousness was restored, often requiring mechanical restraint; dilated pupils, and right radial pulse fuller and stronger than the left; the urine was retained and the right hand and wrist were paretic. There was marked aphasia—*e. g.*, the patient said "talp that" for stop that, "guth Got" for good God, and "15 Avenue B" when asked his name. The difference in the fullness and strength of the radial pulses continued to be strongly marked at all times till death on the eighteenth day. The dilatation of the pupils, which remained sensitive till the seventeenth day, was also permanent. Delirium persisted, and speech was infrequent and unintelligible till the close of the first week; the mental condition then became brighter and speech distinct and coherent, but delusions were constant and the patient was at no time able to recognize his family or friends. There were subsequent alternations of restlessness and excitability with somnolence or lethargy, but no cessation of delirium, delusions, and more or less incoherent and unintelligible speech, till final unconsciousness, which occurred three days before death. Sensitiveness to external irritations was marked throughout this later stage. The control of urine and fæces was permanently lost during the first few days. The paresis of the right hand was much diminished during the first week. On the sixth day, and on the seventh, there was a short, severe, convulsive attack, followed by a transient high temperature. These were succeeded on the morning of the eleventh day by a general convulsion, which was at first confined to the upper extremities, and continued twenty minutes; the right arm was less rigid than the left. Another attack in the afternoon of the same day, of twenty-five minutes' duration, began with a twitching of the facial muscles, and was extended to the trunk; all the extremities remained rigid; the face was of a natural color, though subsequently much flushed, but the hands were blue. The morning convulsion was followed by prolonged unconsciousness, that of the afternoon by an apparently natural sleep after a short interval in which the mind was unusually clear and alert. There was another very brief general convulsion five days later. Posterior cervical muscular rigidity existed from the ninth to the fourteenth days. The temperature on admission was 101·8°, and varied from 100°+ to 101°+ till the fourth day, when, without other change in symptoms, it rose to 104°; and in the twelve hours following declined to 101°+, and was continuous at about that degree till the tenth day, except at the time of the first and second convulsive attacks, when it rose for a short time to 106° and

106·6°; on the morning of the tenth day it rose to 105°, again declined to 101°, and with the occurrence of the third and fourth paroxysms on the eleventh day it rose to 105·4°; on the twelfth day it declined for a brief interval to normal, and was subsequently uniformly high, from 103° to 106°, and at death was 108°. In fifty-two observations the right axillary temperature was higher than the left in thirty-two, the left higher than the right in seven, and in thirteen the two were uniform; the variation was from two tenths of a degree to a degree and eight tenths. The pulse on admission was 112, and then, for the first ten days, 65 to 100; never afterward below 120. The respiration on admission was 36, and after the first four days rarely below 32.

CASE XXII.—Male, aged forty-two years; fell in the street, striking the back of his head; consciousness lost, but regained on the way to the hospital; mental condition stupid, but rational, becoming normal in a few hours; slight general headache; later, frontal pain, followed same day by a single general convulsion of five minutes' duration; head and eyes turned to the right; left side and extremities actively convulsed; right arm and leg motionless. On the third day there was transient posterior cervical rigidity, and on the third and fourth days the left radial pulse was fuller and stronger than the right. During the first ten days the patient's condition was marked by stupor, occasional somnolence, slowness or refusal to answer when questioned, nocturnal delirium becoming continuous, frontal pains, and contracted pupils. In the week following there were delusions, lack of fæcal and urinary control, increased somnolence and stupor, some muttering delirium, and pains in the back of the head and left extremities, succeeded by left paresis. After this time the patient occasionally indicated more intelligence when roused from his habitual stupor, and once conversed intelligently with his wife. The pupils remained contracted and insensitive to light, the urine and fæces uncontrolled, the limbs drawn upward, and any disturbance of the left side of the body was resented. On the twenty-fifth, the last day of life, articulation was indistinct, deglutition difficult, and death, preceded by restlessness and some brightening of the mental condition The temperature on admission was 98°, rose to 103·2° on the third day, and was subsequently 99° to 100°+ till the last day, when it was 107·2°. The pulse on admission was 80, on the fifth day 42 to 58, and at other times 68 to 100. The respiration varied from 18 to 24. A few hours before death both pulse and respiration became frequent.

Case XXIII.—Male, aged thirty-eight years, admitted in an alcoholic condition without a history; profuse hæmorrhage from the left ear. The patient never afterward remembered having been hurt. During the first week hearing was greatly impaired in both ears, and there was much mental confusion, with sensory aphasia and general loss of memory. General headache was severe and vertigo marked. The patient was enabled to recollect with great difficulty the place of his employment, and could only suggest his occupation as a waiter by using an imaginary corkscrew in dumb show. The right radial pulse on the second day was fuller and stronger than the left. In the second week hearing was quite restored in the right ear and was nearly recovered in the left. The mental condition became normal, and there were no further symptoms. The temperature on admission was 99·2°, and did not subsequently exceed 100°+. The left axillary temperature was two tenths of a degree higher than the right when there was a lack of symmetry. The pulse on admission was 80, and was only once above 100. The respiration was from 18 to 24.

Case XXIV.—Male, aged forty years; fell six feet into an area way; consciousness lost, and not restored at time of admission, but sensitiveness to external impressions retained; profuse hæmorrhage from right ear; left radial pulse fuller and stronger than the right; pupils contracted, but responsive to light; right corneal reflex diminished; right side and right face paretic, and urine retained. The pupils became normal on the second day, and the radial pulses symmetrical on the third, with some signs of returning consciousness. Convulsive movements of the extremities occurred on the fourth day, and there was some dysphagia. The patient gave little evidence of intelligence till the end of the fourth week; he had no power of speech beyond the utterance of an occasional single word, and when his attention could be attracted, which was not often, replied only in inarticulate sounds; he rarely recognized his immediate family, and had no apparent comprehension of what was said to him. The right facial paralysis continued, with added ptosis of the left eye, and both pupils became dilated. His mind then became clearer, but intelligence was very limited; he articulated several words with moderate distinctness, and a little later used several short phrases with propriety; a little later still his attention could be momentarily fixed to comprehend and answer monosyllabically a simple question. An ophthalmic examination made by Dr. Callan disclosed no retinal changes. At the end of the sixth week he began to notice what went on about him, recognized his mother, and developed destructive tendencies. Early in the seventh week he first gave attention to the natural offices of the body, and his increasing range of words accentuated his aphasia. After the second month there was only a trace of facial paralysis, and no other paretic condition. He could dress himself, and went about the ward; he could remember, and could write, his name and address correctly, and seemed to readily understand such questions as were asked him, but replied in an endless tirade which was incoherent and largely made up of inarticulate sounds interspersed with recognizable words, and apparently as devoid of meaning to himself as to the listener. He was unable to write from dictation more than a few words before the written characters became incomprehensible, and he repeated words. He had no knowledge of his occupation, manner of injury, or local surroundings. He was discharged at the end of the third month, and had then upon cursory examination no symptoms of mental disorder remaining, except some hesitancy in collating words, and in long sentences a little confusion in expression. If an attempt was made, however, to engage him in a sustained conversation, his thoughts became more and more entangled; he talked rapidly and excitedly, and his words were inextricably jumbled together. On examination, four months later, his mental condition was that of dementia.

The temperature on admission was 98·4°, and reached its maximum, 102·6°, on the second and third days. It then gradually but irregularly declined. It was occasionally normal after the first week, but ordinarily 99°+ or 100°, quite up to the time of the patient's discharge from the hospital. The right axillary temperature was the higher twenty-two times, and the left twelve times, and the two were uniform once, in thirty-five observations made during the first nine weeks. The left was afterward usually two tenths to four tenths of a degree the higher. The pulse did not exceed 90 after the fourth day, and the respiration was at no time more than 22.

Case XXV.—Male, aged forty years; fell two stories from a fire escape to the pavement below; was conscious and delirious when seen by the ambulance surgeon. There was a small linear wound in the left antero-inferior parietal region, hæmorrhage from mouth and nose, and lack of urinary control. Mild delirium and great restlessness continued for twenty-four hours, and the right side and extremities were noted to be warmer and in more active motion than the left. On the second day there was post cervical rigidity, and the patient became more difficult to rouse. On the third day both pupils were somewhat dilated, the face was flushed, and he lay motionless, with eyes closed, irresponsive to questions or to irritations. On the fourth day post-cervical rigidity disappeared. On the sixth day urinary and fæcal control was regained. On the seventh day the patient, fully aroused from his condition of stupor, became restless, and was delirious through the night, but not afterward. From this time he suffered only from mental disorder. He had confusion of ideas and failed to recollect any of the circumstances which preceded his injury, or in fact that he had received a hurt. He had no appreciation of his surroundings, and gave fanciful explanations of his presence in a hospital when interrogated. His mind was alert and his speech coherent. During the second month he suffered an attack of facial erysipelas upon the side opposite the original wound, and was actively delirious. His mental processes were afterward slow, and when questioned he remained long buried in thought before making answer, which when made, though hesitating, was fairly intelligent. He had come to realize that he was in a hospital, but was still ignorant how he happened to be there; "supposed" he had been hurt. The temperature on admission was 97·4°, rose during the day to 102·2°, and was afterward usually from 99°+ to 100°+. The right axillary temperature was two tenths of a degree to a degree higher than the left during the first week, and the left the slightly higher of the two after that time. The pulse and respiration were practically normal at all times.

Case XXVI.—Female, aged twenty eight years; gunshot wound through right temporal region inflicted during a paroxysm of suicidal mania; ball of thirty-two caliber; primary unconsciousness. On admission three hours later no general symptoms; wound of entrance, half an inch posterior to right external angular process, Y-shaped, three quarters of an inch in length in each of its arms; surface powder-stained, some grains of powder imbedded in the substance of the temporal muscle, but none in the skin; profuse hæmorrhage had occurred from the wound and still continued from the mouth and nose; bullet entrance through the bone small and circular and covered by a valve of muscular tissue. On examination the patient was fully conscious, rational, and self-possessed. The right eye was swollen, the lids ecchymotic, and vision on that side entirely lost. The ball had passed from the temporal fossa beneath the lesser wing of the sphenoid and through the floor of the middle fossa at the margin of the sphenoid body. The track was easily followed through the anterior cerebral lobe, and the bony margin of exit could be defined by slightly opening the blades of the short bullet forceps which had been inserted. A small portion of brain matter, not larger than a pea, escaped from the external wound. On the following day she was rather stupid, and another trivial amount of brain matter was extruded. On the third day the left eyelids became moderately ecchymotic and the right side of the face and neck much swollen and painful. There were convulsive movements of the hands and feet, and a loss of smell in the right nostril was confirmed by careful examination. On the fourth day she was quiet and somnolent and had some headache. The left side of the mouth was drawn a little upward and tenderness existed behind the left ear. On the fifth day somnolence and headache ceased, pain and swelling of the right side of the face and neck diminished, and the mental condition became brighter. On the tenth day she was restless and began to suffer pain on the right side of the head, which, on the succeeding day, was intense. The eye became more vascular, swollen, and prominent, and on the fourteenth day was extirpated under ether. At the end of a month the bullet wound of entrance had become simply cutaneous and was in process of cicatrization. The swelling of the right side of the face and neck and the tenderness behind the left ear had ceased to exist. The pain on the right side of the head, which persisted in some degree, was no longer constant or the source of any considerable discomfort. There had been no indication of any form of mental impairment at any time since the slight hebetude on the second and third days after the reception of injury, and no loss of fæcal and urinary control.

The temperature on admission was 100°; rose to 102·4° in twelve hours, and declined to 99·8° on the second day, and then varied from 101°+ to 99°+ till the twelfth day; it did not exceed 100° after the fifteenth day and was subsequently from 99° to 100°. The right axillary temperature was habitually two tenths of a degree higher than the left. The pulse was from 72 to 80 till the third day, from 68 to 52 till the twelfth day, and subsequently from 70 to 78. The respiration was 28 on admission and afterward normal—16 to 22.

In the sixth week the wound had healed and there were no symptoms. At the end of three months her mental and physical condition is normal, in her own opinion better than before the injury was received.

## SUMMARY.

The differential diagnosis of the intracranial lesions has been incidentally established in the consideration of their individual symptoms. There are few instances in which the nature of the essential lesion and of its complications can not be determined with substantial certainty, and in a considerable proportion of cases its location even can be fixed with some approach to precision. The fact that an interval of time may be required for the evolution of symptoms is paralleled in the case of idiopathic diseases affecting the great cavities of the body ; it can scarcely be considered a special diagnostic difficulty, therefore, in the present class of traumatisms. In lesions of the parenchyma the delay is unimportant ; in hæmorrhages of the form in which promptitude in diagnosis is demanded by the necessity for promptitude in action, the development of the case is likely to be correspondingly rapid and decisive.

The existence of superficial injuries of the head, the evidences of fracture, and the elevation of temperature, individually or collectively, together with the usual processes of diagnostic exclusion, will be sufficient to determine the fact that some encephalic injury has been suffered. The further determination of the special lesion which dominates the case presents difficulties which, while not insuperable, are often considerable. It is perhaps useless to attempt a more condensed summary of points in differential diagnosis. I have already incidentally stated them as succinctly as seemed compatible with their proper presentation ; but it may be of service to recall, or to reiterate, some of the more important diagnostic indications which the study of symptoms has suggested.

*Hæmorrhages.*—The morbid conditions which may directly result from traumatic intracranial hæmorrhages are : an abnormal temperature, a complete or partial loss of consciousness, a change in the character or frequency of the pulse or respiration, a disturbance or abrogation of muscular function, and an irregularity of the pupils. These conditions are subject to complication, modification, or supersedure by the symptoms of coexistent lesions.

A continued subnormal temperature is characteristic of large and comparatively uncomplicated hæmorrhages, and as these are more frequently of epidural character, it may be regarded as to a certain extent diagnostic of the variety as well as of the class. The absence of symptoms indicative of parenchymatous injury will be confirmatory of the opinion that an existent hæmorrhage is derived from the epidural vessels. Associated symptoms of diffused contusion suggest a pial, and those of laceration a cortical, hæmorrhage. In the majority of cases the primary record of temperature is from 99° to 99°+, and in any case in which, then or afterward, it exceeds 101°+, or probably 100°, the elevation is due to an associated lesion. It follows that in pial or cortical hæmorrhages the temperature has a higher range than in those of epidural origin, and is proportionate to the extent and importance of the complication. The bilateral variation to which the axillary temperatures are subject is not peculiar to this result of injury.

The primary unconsciousness which is of frequent occurrence in cases of hæmorrhage is a symptom of complicating general contusion; the secondary unconsciousness, due to the loss as well as pressure of blood effused, follows with or without an interval of restored consciousness, dependent upon the severity of the diffused injury of the parenchyma and the rapidity of the hæmorrhagic effusion, and is partial or complete in proportion to its amount. Consciousness is always lost in fatal cases; it is retained in fifty per cent. and more in recovering cases, even in those demanding operation.

The character and frequency of the pulse have no definite relation to the form, location, or amount of hæmorrhage. The pulse may be normal, slow, or frequent in large extravasations wherever situated; but frequency is of so much more usual occurrence in hæmorrhage than in other intracranial lesions that when noted it may be considered fairly diagnostic, with the numerical probabilities in favor of its epidural character. The bilateral variation in the force and fullness of the arterial pulsations is common to hæmorrhages and to injuries of the brain substance, and of importance, therefore, only in general diagnosis.

An alteration in the character or frequency of respiration is almost invariable in fatal cases in which hæmorrhage is an approximately isolated lesion. When the effusion is upon the convex surface of the brain, respiration is usually frequent and often stertorous; when at the base posteriorly, it may be frequent with cyanosis, or, if pressure is made upon the medulla, it becomes progressively slower until it ceases altogether, though cardiac and arterial pulsation may still continue. In recovering cases it is habitually unchanged. In complicated or mixed cases it, like the pulse, perhaps as a resultant of opposing forces, very generally remains normal; and if abnormal it is more likely to be stertorous than unduly slow or frequent.

General or local paralysis and disordered muscular action may be direct symptoms of hæmorrhage compressing or irritating recognized centers of muscular control; tetanic spasm is not infrequent, but clonic contractions are of rare occurrence, except as the result of an associated lesion.

The pupillary condition usually suffers some change, but none which is characteristic. Every possible combination of contraction, dilatation, and normal condition, with the single exception that contraction of one pupil never occurs without some change in its fellow, is associated with every variety and situation of hæmorrhage. Dilatation in some combination is more commonly observed than contraction, but not more frequently upon the side of the effusion than upon the opposite; and not more characteristically with one type of hæmorrhage than with another. In complicated hæmorrhages dilatation of both pupils is more common, and the effusion is more frequently bilateral than in the more nearly simple cases; and in unilateral dilatation is more likely to be on the corresponding side. A normal condition of the pupils is compatible with every variety of hæmorrhage wherever situated, whether simple or complicated.

Sensory disturbances, as delirium or irritability, are not symptoms of hæmorrhage, and when they occur are to be regarded as indicative of an accompanying lesion of the parenchyma.

*Subarachnoid Serous Transudation.*—The serous transudation from the pial vessels which occasionally results from meningeal contusion can not be connected with symptomatic conditions.

*Arachnitis* is either acute or subacute in form, and is typically caused by a diffused meningeal contusion, though exceptionally propagated from a point of localized injury. It is sometimes an immediate result of the meningeal lesion, and it may be insidious in its inception and progress, but its beginning is usually late and is sharply defined. Its invasion is likely to be marked by a distinct and rather sudden elevation of temperature and an evident change in the general condition of the patient. The subsequent course of temperature is erratic, and the characteristic symptoms are those of cortical irritation. The pupils are oftener normal than otherwise, and changes in the characters of the pulse and respiration are slight. The form of the effusion is not necessarily reflected in the course and nature of the symptoms. The question of infection is un certain.

*General contusion* is a constant complication of all other forms of intracranial injury, but rarely occurs as an isolated lesion of fatal severity. Its symptoms are irregular in their development, course, and termination, and indefinite in their mutual relation. This lack of conformity to any classical rule is due to the comprehensiveness of the lesion, its regional variations, and the fluctuations which occur from time to time in the distribution of the movable fluids upon which its manifestations mainly depend. A loss of consciousness, at some time and in some degree, is more nearly constant than any other individual symptom, and the conditions of temperature are more uniform than any of the other phenomena which it occasions. The temperature is not likely to be subnormal at the time of earliest observation, nor to exceed 99° + ; its subsequent course in cases of intensity is progressive, with few recessions, and ultimately reaches elevations of high degree.

Primary or early delirium, like primary unconsciousness, in both simple and complicated cases, is to be ascribed solely to the influence of this lesion. The diagnosis must largely depend upon the recognition of the fact of intracranial injury, and upon the further possibility of excluding its other varieties, or, if they exist, of segregating the effects which they produce from a distinct remainder of symptoms.

*Limited contusion* is comparatively infrequent, and when it occurs in scattered areas through the centrum ovale, is not distinguishable from the general form of the same lesion; when it is cortical, it differs from laceration only in the extent of injury done to tissue; and symptoms, if they result, differ only in degree. It is therefore practically impossible to diagnosticate it from those lesions in their mitigated form.

*Laceration* is almost, if not quite, invariably complicated by a concomitant general contusion and by a resultant hæmorrhage. The primary loss of consciousness, and the delirium of some grade or character which often precedes or follows its restoration, are attributable to the attendant general contusion. In trivial cases there may be no secondary symptoms which indicate the fact of laceration. The primary unconsciousness may be replaced by a condition of lethargy or blunted perception, passing through somnolence into coma and death. The primary stage is most frequently succeeded by mental aberration or decadence, which may terminate in recovery, permanent dementia, or death. In exceptional instances consciousness may remain unimpaired, with extensive laceration of even fatal import. There is no necessary relation between the gravity or simplicity of the early psychic symptoms and the outcome of the case.

The temperature is higher than in any other form of intracranial injury, and, in cases destined to an early fatal termination, is characterized by a rapid and progressive increase, which sometimes continues for a certain time after death has occurred.

An irritability or abnormal sensitiveness to external impressions, often noticeable even after the supervention of final unconsciousness, and wanting in cases of hæmorrhage or contusion, is of frequent occurrence.

Convulsions, especially in implications of the frontal or temporo-sphenoidal lobes, are frequent in fatal cases, and so infrequent in the history of other lesions that they may be regarded as characteristic. The presumption that they are occasioned by laceration rather than by hæmorrhage is strengthened by a previous high temperature.

The loss of fæcal and urinary control is common to all extensive lacerations without reference to the abrogation of consciousness or of muscular power. It rarely follows other forms of intracranial injury and is very nearly pathognomonic. The urinary and fæcal discharges may be either unconscious or involuntary, or they may be the result of the patient's indifference to his surroundings. There are no demonstrated centers of control.

Paralyses are so much oftener the result of other lesions that they are of service only in determining the location of a laceration the existence of which has been already predicated upon more positive manifestations.

The pupillary changes have no greater diagnostic value than in hæmorrhages; the pupils are, in fact, normal in a much larger proportion of cases.

The characters of the pulse and respiration are habitually unchanged unless modified by the existence of complications. The contrast afforded by their substantially normal condition in an environment of pathic phenomena gives them the highest diagnostic value which they possess in this particular relation.

The bilateral variation in axillary temperatures and in the force and fullness of arterial pulsation, already noted as of unknown origin and referred to general diagnosis, is common to all forms of intracranial injury.

The manifestations of psychic disturbance are confined to cases in which the frontal lobes are implicated, but this implication is so constant as to make them practically symptoms of laceration in general. The other special symptoms which localize the seat of laceration have been already summarized.

The phenomena as indicated which directly point to laceration may be enumerated as certain peculiarities of temperature, psychic disturbances, loss of fæcal and urinary control, and clonic convulsions.

*Pyogenic parenchymatous inflammation* is infrequent, and is of limited form, except when caused by the intrusion of a foreign body. Direct laceration and infection through the medium of compound fracture affords no question of diagnosis and is excluded from consideration.

The predisposing cause of traumatic central abscess is limited contusion; the exciting cause is supposed to be the admission of a pyogenic germ from some source external to the body. Though this supposition as to the source of infection may be correct, the further proposition that a route of entrance is always afforded by a superficial wound of the head is erroneous. Cases have occurred and are recorded in which no such wound existed.

The number of instances in which the histories of these limited pyogenic processes have been carefully observed or recorded is insufficient for the formulation of rules for diagnosis. The two cases which I have presented, and a third which I have noted, are in evidence of their uncertain symptomatology.

These conclusions are derived solely from the analysis of the cases which I have detailed, and are stated in as positive terms as the limited number of observations made will warrant. The series of cases presented, if insufficient to afford a basis for statistical inference, is yet so extended that the generalizations which it justifies are entitled to credence until controverted by results obtained from the study of a very much larger number of cases subjected to equally careful examination. It may be questioned whether deductions made, as in this instance, from the comparison of some hundreds of cases are likely to be materially changed by any subsequent multiplication of their number.

Symptoms are so diversified, their combinations so varied, and their continuance is sometimes so brief, that constantly careful observation and equally careful record are essential to thorough comprehension of intracranial injuries. If there are few symptoms which are intrinsically pathognomonic there are many which by mutual relations of time and circumstance assume a pathognomonic character.

The possible multiplicity of lesions must be recognized, the relative as well as the absolute value of symptoms estimated, and if necessary some interval of time afforded for the development of the pathic condition; diagnosis becomes then neither more difficult nor more uncertain than in a majority of grave traumatic or idiopathic lesions.

34 West Thirty-seventh Street.

# The New York Medical Journal.

*A WEEKLY REVIEW OF MEDICINE.*

EDITED BY

FRANK P. FOSTER, M.D.

---

THE PHYSICIAN who would keep abreast with the advances in medical science must read a *live* weekly medical journal, in which scientific facts are presented in a clear manner; one for which the articles are written by men of learning, and by those who are good and accurate observers; a journal that is stripped of every feature irrelevant to medical science, and gives evidence of being carefully and conscientiously edited; one that bears upon every page the stamp of desire to elevate the standard of the profession of medicine. Such a journal fulfills its mission—that of educator—to the highest degree, for not only does it inform its readers of all that is new in theory and practice, but, by means of its correct editing, instructs them in the very important yet much-neglected art of expressing their thoughts and ideas in a clear and correct manner. Too much stress can not be laid upon this feature, so utterly ignored by the "average" medical periodical.

Without making invidious comparisons, it can be truthfully stated that no medical journal in this country occupies the place, in these particulars, that is held by THE NEW YORK MEDICAL JOURNAL. No other journal is edited with the care that is bestowed on this; none contains articles of such high scientific value, coming as they do from the pens of the brightest and most learned medical men of America. A glance at the list of contributors to any volume, or an examination of any issue of the JOURNAL, will attest the truth of these statements. It is a journal for the masses of the profession, for the country as well as for the city practitioner; it covers the entire range of medicine and surgery. A very important feature of the JOURNAL is the number and character of its illustrations, which are unequaled by those of any other journal in the world. They appear in frequent issues, whenever called for by the article which they accompany, and no expense is spared to make them of superior excellence.

---

**Subscription price, $5.00 per annum. Volumes begin in January and July.**

---

PUBLISHED BY

D. APPLETON & CO., 72 Fifth Avenue, New York.

LANE MEDICAL LIBRARY

To avoid fine, this book should be returned on
or before the date last stamped below.

93 Phelps, C. 15677
53 The differential diag-
895 nosis of traumatic intra-
cranial lesions

NAME DATE DUE